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To my parents

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ABSTRACT

Ageing, the phenotypic and physiological changes with age, is widespread across taxa. Yet, the patterns of ageing and lifespan vary remarkably both within and among species. Understanding the sources of this diversity remains a central challenge in evolutionary biology, particularly in natural populations exposed to fluctuating environments. In this thesis, I used exceptionally detailed long-term data from two long-lived vertebrates, the Asian elephant (*Elephas maximus*) and the Alpine swift (*Apus melba*), to investigate the determinants of ageing diversity in the wild. I first determined the age-related changes in biometric traits, reproductive traits, and parasite load in the Alpine swift, revealing clear senescence across multiple traits, but also pronounced asynchrony in the onset and rate of senescence between traits and between sexes. Then, I investigated the transgenerational effects of senescence by examining how parental age at conception shapes offspring life histories in the same population. I revealed sex-specific effects: while older mothers and fathers produced larger offspring before fledging, older fathers produced sons with higher parasite loads and shorter lifespans. Finally, I investigated the genetic basis of lifespan and survival variation in the Asian elephant population. I found substantial additive genetic variance and heritability for lifespan in both sexes, and for survival early in life (0 to 5 years old), which declined to near zero thereafter. I also found low cross-sex genetic correlation, suggesting sex-specific genetic architecture for lifespan. Across all chapters, environmental conditions emerged as a major modulator of ageing. Adverse early-life environments in Alpine swifts and spatiotemporal environmental variation in Asian elephants each explained significant variation in lifespan. Overall, this thesis provides an integrative perspective on ageing in the wild, demonstrating that ageing diversity arises from the combined influence of genetic factors, parental effects, life-history strategies, sex differences, and environmental conditions experienced across the lifespan.

KEYWORDS: Ageing, Alpine swift, Asian elephant, Heritability, Life-history, Lifespan, Long-lived species, Parental age effects, Quantitative genetics, Senescence.

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TIIVISTELMÄ

Vanheneminen, joka tuo mukanaan fenotyyppisiä ja fysiologisia muutoksia, on laajalle levinnyt biologinen ilmiö eri taksoneissa. Ikääntymisen kulku ja elinikä vaihtelevat kuitenkin huomattavasti eri eliöryhmissä sekä lajien välillä että niiden sisällä. Ikääntymisen vaihtelua selittävät tekijät ovat keskeisiä evoluutiobiologisia kysymyksiä erityisesti ympäristöolosuhteiltaan vaihtelevissa oloissa elävissä luonnonpopulaatioissa. Hyödynnän väitöskirjassani kahdesta pitkäikäisestä selkärankaisesta, aasiannorsusta (*Elephas maximus*) ja alppikiitäjästä (*Apus melba*), olevia poikkeuksellisen yksityiskohtaisia pitkittäisaineistoja tutkiakseni ikääntymisen monimuotoisuuden taustalla olevia tekijöitä. Ensimmäisessä osatyössä tarkastelin ikääntymisen aiheuttamia muutoksia alppikiitäjän biometrisissä muuttujissa, lisääntymisessä ja loisten määrässä. Monet tutkitut piirteet muuttuivat iän myötä, mutta ikääntymisen alkamisajankohta ja etenemisnopeus vaihtelivat paljon eri piirteiden ja sukupuolten välillä. Seuraavaksi tutkin ikääntymisen ylisukupolvisia vaikutuksia analysoimalla, miten alppikiitäjävanhempien ikä hedelmöityksen hetkellä heijastuu jälkeläisten elämänkulkuun samassa populaatiossa. Sukupuolet erosivat toisistaan siten, että vaikka sekä vanhemmat äidit että isät tuottivat poikasia, jotka olivat suurempia ennen pesästä lähtöä, vanhemmat isät tuottivat poikia, joilla oli enemmän loisia ja jotka kuolivat nuorempina. Lopuksi tarkastelin eliniän ja elossapysymisen periytyvyyttä aasiannorsuyhteisössä. Additiivinen geneettinen vaihtelu ja periytyvyys selittivät merkittävästi koko eliniän pituutta sekä varhaiselämän (0–5 vuoden iässä) eloonjäämistä molemmilla sukupuolilla, mutta vaikutukset vähenivät tämän jälkeen lähes nollaan. Sukupuolten välinen geneettinen korrelaatio oli heikko, mikä viittaa urosten ja naaraiden erilaiseen eliniän geneettiseen säätelyyn. Kaikissa osatöissä merkittävä ikääntymistä muokkaava tekijä olivat ympäristöolosuhteet. Alppikiitäjillä epäsuotuisat olosuhteet elämän alussa ja aasiannorsuilla ajallisesti ja paikallisesti vaihtelevat ympäristöolot selittivät merkittävän osan eliniän pituuden vaihtelusta. Väitöskirjassani osoitan, että luonnonpopulaatioiden ikääntymisen moninaisuus koostuu geneettisten tekijöiden, vanhemmuuden vaikutusten ja ympäristöolosuhteiden yhteisvaikutuksesta.

ASIASANAT: Ikääntyminen, Alppikiitäjä, Aasiannorsu, Periytyvyys, Elinkiertostrategiat, Elinikä, Pitkäikäiset lajit, Vanhempien iän vaikutus, Kvantitatiivinen genetiikka, Senesenssi.

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Abbreviations

AFR	Age at first reproduction
DNA	Deoxyribonucleic acid
GLMM	Generalized Linear Mixed Model
h^2	Narrow-sense heritability
LMM	Linear Mixed Model
LRS	Lifetime reproductive success
MTE	Myanma Timber Elephants
SE	Standard Error
SD	Standard Deviation
V_A	Additive genetic variance
V_{cohort}	Variance from the 5-year cohort
V_M	Maternal variance
V_P	Phenotypic variance
V_R	Residual variance
V_{region}	Variance from the regions in Myanmar
r_A	Cross-sex genetic correlation

List of Original Publications

This dissertation is based on the following original publications, which are referred to in the text by their Roman numerals:

I **Moullec H.**, Reichert S., and Bize P.; Aging trajectories are trait- and sex-specific in the long-lived Alpine swift. *Frontiers in Ecology and Evolution,* 2023; 11. https://doi.org/10.3389/fevo.2023.983266

II **Moullec H.**, Berger V., Meier C., Reichert S., and Bize P. Effects of parental age at conception on offspring life history trajectories in a long-lived bird. *Evolution,* 2025; qpaf181. https://doi.org/10.1093/evolut/qpaf181

III **Moullec H.**, Lummaa V., Htut W., Min Oo Z., Berger V., Briga M., Reichert S. Additive genetic variance reveals the evolutionary potential of longevity in a long-lived mammal. *Submitted Manuscript.*

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1 Introduction

1.1 The study of ageing and senescence

Ageing is defined as the progressive phenotypic and physiological changes that occur in an organism throughout its life. Senescence, more specifically, refers to the gradual physiological and functional decline of an organism with increasing age, manifested by a loss of function, decreased reproductive success, and increased mortality (Bronikowski & Flatt, 2010; Rose, 1991). Although ageing can refer to phenotypic changes that do not necessarily impair performance, such as the whitening of hair at advanced ages in mammals (Caro & Mallarino, 2020), ageing and senescence are often used as synonymous in the literature for simplicity (Monaghan et al., 2008). In this thesis, both terms will likewise be used synonymously.

Because natural selection should favour long survival and reproduction, why selection cannot prevent ageing is often seen as an evolutionary paradox. Understanding why senescence persists is therefore a central challenge in evolutionary biology (Monaghan et al., 2008). The study of senescence has evolved significantly over the past two decades (Gaillard & Lemaître, 2020). For a long time, senescence was believed not to occur in wild populations, as organisms in nature face multiple hazards (e.g., competition, predation, accidents, pathogens) and were therefore unlikely to survive long enough to be affected by senescence (Medawar, 1952). Thanks to increasing longitudinal studies and monitoring of life-history traits of known-age individuals throughout their lives (Clutton-Brock & Sheldon, 2010), there is now extensive evidence of actuarial senescence (i.e., increase in mortality rate/decrease of survival probability with advancing age) and reproductive senescence (i.e., the decline in reproductive success with age) in wild vertebrates (Gaillard & Lemaître, 2020; Lemaître, Ronget, & Gaillard, 2020; Lemaître & Gaillard, 2017; Nussey et al., 2013). Since it is now established that senescence is widespread throughout the animal kingdom, the new challenge is to determine whether senescence is affecting all traits, whether senescence patterns (onset and rate of senescence) are similar across traits and individuals, and identify the factors involved in ageing diversity among and across species (Gaillard & Lemaître, 2020).

1.2 The diversity of ageing

1.2.1 Among-species diversity of ageing

Species in the wild exhibit striking variation in lifespan. Some species can live well over a century; for example, the Galapagos tortoise (*Chelonoidis nigra*) can reach ages of up to 177 years (Hoekstra et al., 2020). More extreme cases are also reported, like the Greenland shark (*Somniosus microcephalus*), with individuals estimated to live at least 272 years (Nielsen et al., 2016). On the other hand, some species are extremely short-lived, such as mayflies (order *Ephemeroptera*), which adult stage (after metamorphosis) lasts from one hour to a few days until death (Brittain, 1982). Interestingly, although senescence was evidenced to be widespread in the wild, some modular organisms, as opposed to unitary organisms, seem not to be affected by it (Bernard et al., 2020), such as the freshwater polyp (*Hydra vulgaris*). This absence of senescence in Hydra would rely on an indefinite and continuous self-renewal of stem cells, allowing a continuous replacement of tissues (Khokhlov, 2014; Schaible et al., 2014; Schenkelaars et al., 2020), and the anatomically separated modules isolating the deterioration and mortality risks, thus preventing consequences for the rest of the organism, as opposed to unitary organisms (Bernard et al., 2020). In mammals, comparative studies found that larger species, with greater body mass, tend to experience lower mortality rates, delayed senescence, and longer lifespans compared to smaller species (Péron et al., 2019). Interestingly, for the same body mass, birds show longer lifespans than mammals, although they have higher metabolic rates, glycaemia, and body temperature (Holmes & Austad, 1995; Moreno Borrallo et al., 2025; Speakman, 2005). In addition to these enormous differences in lifespan, there is accumulating evidence of species differences in the rates of actuarial senescence, as well as in the rates of senescence of multiple physiological and phenotypic traits (Bouwhuis & Vedder, 2017a; Gaillard & Lemaître, 2017). For instance, a comparative study across 46 species from different taxa found substantial diversity in both the direction and magnitude of age-related changes in traits such as mortality and fertility (Jones et al., 2014).

1.2.2 Inter-individual diversity of ageing

At the intraspecific level, individuals are not equal when facing the ageing processes. There is a high inter-individual variation in ageing patterns (i.e., onset, timing, and rate). Some individuals live longer, remain fertile and in good health longer, and age more slowly than others. In addition, there are high variations in how the biological age of organisms reflects their chronological age. Chronological age, the time passed since an individual's birth, progresses at a fixed, immutable rate. In contrast, biological age reflects the functional state of an organism and is shaped by intrinsic (e.g., genetics) and extrinsic factors (e.g., environmental stressors), modulating its progression rate (Jylhävä et al., 2017; Ries & Pöthig, 1984). As a result, individuals of the same chronological age can end up with different biological ages (Jylhävä et al., 2017). These inter-individual differences are at the core of ageing studies in humans, which aim at understanding the ageing mechanisms to improve healthy ageing and extend human lifespan.

Ageing affects a large range of traits from the phenotypic level (e.g., survival, reproduction, behaviour; Lemaître, Ronget, & Gaillard, 2020; Monaghan et al., 2008; Nussey et al., 2013), to the physiological and cellular level (e.g., decrease of telomere length, increase of oxidative damage; Monaghan et al., 2009; Monaghan & Haussmann, 2006) through which the inter-individual diversity of ageing can arise. More generally, studies in biogerontology have identified twelve mechanisms, known as the hallmarks of ageing (López-Otín et al., 2023), that modulate the rate of ageing and underlie the age-related phenotypic changes observed throughout the life of an organism. This thesis focuses on the phenotypic changes associated with ageing, while drawing on these underlying mechanisms to discuss and interpret some of the patterns observed.

1.2.3 Asynchrony of ageing between traits

Initially, studies on ageing primarily focused on actuarial senescence (decline of survival probability) and the senescence of reproductive traits (Bouwhuis & Vedder, 2017b; Lemaître, Ronget, & Gaillard, 2020; Lemaître & Gaillard, 2017; Nussey et al., 2013). This bias in single-trait limits our understanding of senescence (Lemaître & Gaillard, 2017). However, since the 1990s, there has been an increasing number of studies investigating the senescence of other physiological and phenotypic traits such as behaviour, morphology, and immunity (Nussey et al., 2013). Studies on natural populations found, for example, evidence of body mass decline at old ages in ungulates (Hayward et al., 2015; Nussey et al., 2011; Weladji et al., 2010), changes with age in foraging behaviour in albatrosses (Catry et al., 2006; Lecomte et al., 2010), changes of morphology and colour with age in birds (Adamkova et al., 2022a; Evans et al., 2011; Møller & DE Lope, 1999; Palestis et al., 2012; Siefferman et al., 2005), and immunosenescence in various species of birds, mammals and reptiles (Palacios et al., 2011; Peters et al., 2019). In addition to investigating a larger number of traits, studies in the wild directly comparing the differences in senescence rate between these traits are also increasing, which is key to getting a better understanding of the diversity of ageing patterns and their evolution . Importantly, as opposed to what was first believed by evolutionary biologists, that senescence is synchronised across traits (Maynard Smith, 1962; Williams, 1957, 1999), studies in humans and laboratory animals showed evidence of uncoupled senescence between biological functions (Bansal et al., 2015; Burger & Promislow, 2006; Grotewiel et al., 2005; Herndon et al., 2002; Martin et al., 2007; Moorad & Ravindran, 2022; Rueppell et al., 2007). More recently, a growing number of studies in wild populations have evidenced an asynchrony in the onset and rate of senescence across survival and reproductive traits (Bouwhuis et al., 2009; Bouwhuis & Vedder, 2017a; Cooper et al., 2021; Fay et al., 2021), as well as diverse phenotypic traits (Cornwallis et al., 2014; Evans et al., 2011; Hayward et al., 2015; Kervinen et al., 2015; Preston et al., 2011). Because the timing of senescence differs between traits, ageing patterns can be influenced by a cascade of interactions with age between different physiological and phenotypic traits. For example, reproductive senescence may be induced by a decline in foraging performance, which in turn may be triggered by a decline in muscle mass and physical activity. This would translate into an earlier onset of senescence in mass versus foraging ability versus reproduction. However, much remains to be done to understand which traits senesce first and why (Moorad & Ravindran, 2022). Therefore, comparative analyses of the onset and rate of senescence across multiple phenotypic traits are needed to get a thorough understanding of ageing patterns and how they evolved.

1.3 The evolutionary theories of ageing

The mechanism of natural selection, first described by Darwin in 1859, shapes organisms to optimise survival and maximise reproductive success. The persistence of senescence, despite its fitness costs, has thus raised great interest among theorists who have examined the issue in an effort to solve this evolutionary enigma. To answer this paradox and understand why organisms age, several theories have been proposed (Medawar, 1952; Williams, 1957). These theories were developed around the idea that the force of natural selection, which measures how strongly selection influences fitness (survival or fecundity), declines with age (Hamilton, 1966; Hughes & Reynolds, 2005). This came from the realisation that, even in the absence of ageing, the probability of survival and reproduction (i.e., an organism's ability to pass on genes) decreases with age due to extrinsic mortality.

1.3.1 The Mutation Accumulation theory

The Mutation Accumulation theory proposed by Medawar in 1952 suggests that: (i) Individuals are expected to accumulate mutations as they age, which, if deleterious, can impair reproduction and survival at older ages. (ii) Because natural selection strongly declines after individuals have started reproducing, there is little scope for the deleterious mutations to be selected out, leading to their accumulation and a decline in fitness (Medawar, 1952). These harmful mutations would then accumulate via genetic drift in the population and through generations, leading to the evolution of ageing. The reasoning behind the idea that the strength of selection diminishes with age is that, at advanced ages, the contribution to fitness is low because reproduction has decreased or stopped, and survival rates are low. As a result, natural selection to improve fitness would be weak and less effective at eliminating deleterious mutations. Hamilton in 1966 then formalised this theory mathematically by constructing a quantitative model that evaluates how the forces of selection act across an individual's life (Hamilton, 1966).

1.3.2 The Antagonistic Pleiotropy theory

Williams developed Medawar's idea by proposing in 1957 the Antagonistic Pleiotropy theory (Williams, 1957). His theory suggests that the strong force of natural selection early in life may favour alleles with opposite pleiotropic effects, improving survival or reproduction early in life, while possibly exerting harmful effects later in life. Because the force of natural selection is weak at advanced ages, these late-acting deleterious alleles are not affected by selection and thus not selected against. According to Williams, if the benefits early in life exceed the deleterious effects late in life, such alleles would be actively favoured by selection and maintained in the population, leading to the evolution of ageing. Moreover, under Williams' hypothesis, there should be a genetic trade-off between early-life fitness, such as fecundity, and late-life fitness, such as survival. He predicted that genotypes associated with rapid development, leading to earlier reproductive maturity, would also exhibit an earlier onset of senescence. Additionally, he suggested that selection for genotypes with increased early-life fecundity would come at the cost of reduced longevity. Thus, genotypes with high early-life fecundity should be short-lived, whereas genotypes with low early-life fecundity should be long-lived (Hughes & Reynolds, 2005; Rose, 1991; Williams, 1957).

Kirkwood, in 1977, developed a physiological extension to Williams' theory, known as the Disposable Soma hypothesis. It posits that resources available for reproduction and somatic maintenance are limited, leading to a trade-off in their allocation, which is adjusted by natural selection. Investment in long-term somatic maintenance over early reproduction offers little evolutionary advantage, since high extrinsic mortality prevents individuals from reaping the long-term benefits of such investment. As a result, investment in somatic maintenance and repair is lower than what would be required for indefinite survival (Kirkwood, 1977; Kirkwood & Rose, 1991), leading to a progressive deterioration of the soma over time and thus to senescence.

1.3.3 Validation of the theories

Medawar's mutation accumulation theory and Williams's antagonistic pleiotropy theory are the two fundamental evolutionary theories of ageing. Both can shape populations; however, their relative contribution is still unknown (Moorad & Promislow, 2009). Numerous studies on laboratory organisms such as mice, worms, and Drosophila have identified several genes that, when mutated, have an antagonistic pleiotropic effect, decreasing or preventing reproduction while increasing longevity (e.g., daf-2, age-1, chico, prop-1; Austad & Hoffman, 2018). In *C. elegans*, for example, a mutation in the daf-2 gene (a gene which encodes the insulin/IGF receptor, central in the insulin/IGF signalling pathway regulating metabolism, development, and ageing) has been shown to markedly reduce early-life reproduction while substantially extending lifespan (Austad & Hoffman, 2018; Chen et al., 2007; Jenkins et al., 2004; Kimura et al., 1997). Because selection is stronger early in life, these mutants go rapidly extinct when competing against wild-type worms, which display higher early-life reproductive output. In natural populations, quantitative genetic studies also provide support for the main theories of ageing. Several studies have reported an increase in additive genetic variance with age, consistent with the mutation accumulation theory, and a negative genetic correlation between traits associated with early and late-life fitness, supporting the antagonistic pleiotropy theory (Wilson et al., 2008). There is now also extensive evidence of trade-offs between life history traits in wild populations (Stearns, 1989, 1992), often related to the cost of reproduction, which supports the antagonistic pleiotropy theory of aging (Austad & Hoffman, 2018). However, it is still debated whether these trade-offs are primarily driven by antagonistic pleiotropic genes or underlying physiological constraints related to resource allocation (Flatt & Heyland, 2011).

1.4 The life-history theory

Following the development of evolutionary theories of ageing, life-history theory emerged to explain the inter- and intra-specific diversity in survival, growth, and reproduction. Life-history theory is based on the principle that organisms face trade-offs arising from limits in energy allocation that force them to balance investment among competing traits or from genetic constraints. Importantly, these life-history trade-offs are thought to play a central role in shaping the diversity of ageing patterns observed across and within species.

1.4.1 Trade-offs and ageing diversity among species

This interspecific diversity in lifespan and senescence patterns is driven by trade-offs in the allocation of limited resources among life-history traits, such as growth, early-life reproduction, and later-life maintenance and reproductive output (Kirkwood & Rose, 1991; Stearns, 1977, 1992). The life-history trade-off of higher reproductive investment early in life versus stronger late-life senescence, in particular, is predicted by both the antagonistic pleiotropy and disposable soma theories (Kirkwood & Rose, 1991). Selection on life-history trade-offs should optimise an individual's fitness within its environment while being constrained by the organism's structural plan (Bauplan), resource availability, and environmental conditions (Cohen et al., 2020; Gaillard & Lemaître, 2020; Metcalf & Pavard, 2007). In 1977, Stearns formalised the concept of the fast-slow continuum of life histories, which categorises species based on their life-history strategies (Stearns, 1977, 1983). A slow phenotype is associated with a slower growth rate, delayed maturity, longer lifespan, fewer offspring produced that are more dependent on their parental investment early in life, and low adult mortality, allowing more reproductive events throughout life (Stearns, 1992; Stott et al., 2024). Examples of species falling into this strategy are humans (*Homo sapiens sapiens*), Asian elephants (*Elephas maximus*), and blue whales (*Balaenoptera musculus*). In contrast, a fast phenotype is characterised by a faster growth rate, earlier maturity, shorter lifespan, more offspring produced in one reproductive event, and higher adult mortality (Stearns, 1992; Stott et al., 2024). The semelparous Blue-ringed octopuses (*Hapalochlaena lunulata*), for example, fall into this category, as they grow rapidly and die at around 2 years of age after producing numerous offspring in a single reproductive event (Stott et al., 2024). Additionally, species with a fast pace of life are characterised by an earlier onset and faster rate of senescence compared to species with a slower pace of life (Jones et al., 2008). The fast-slow continuum appears to be the main axis describing life-history variation across species, including in birds and mammals (Gaillard et al., 1989; Van de Walle et al., 2023). According to Healy et al., (2019), this fast-slow continuum would explain 70% of the variation in life-history strategies between species. Stott et al., however, highlight that other axes of life-history variation now need to be explored to get a better understanding of the diversity of life histories, for example, taking into account developmental mode and reproductive strategies (Stott et al., 2024).

1.4.2 Trade-offs and ageing diversity between and within individuals

At the intraspecific level, multiple studies examining variation among individuals in ageing in natural populations have provided evidence supporting the life history theory. In particular, they found inter-individual variation in trade-offs between early reproduction and later-life senescence (Bouwhuis & Vedder, 2017a; Nussey et al., 2013). However, individual variation in life-history traits appears to be more complex than the patterns observed across species and does not consistently align with the fast-slow continuum, partly because of the complexity of environmental constraints at the individual level (Van de Walle et al., 2023). For instance, a recent meta-analysis found a positive correlation between survival and reproduction within and between individuals, and highlighted the importance of resource acquisition in shaping life-history trade-offs (Haave-Audet et al., 2022; see section *1.7. Extrinsinc factors affecting ageing*).

Within individuals, trade-offs may also help explain the asynchrony of senescence between traits. In a review of Lemaître et al., (2015), authors surveyed studies testing for an early-late life trade-off across bird, mammal, and reptile populations. Of the 26 studies identified, 21 reported evidence of such trade-offs, involving traits such as early-life reproductive effort and actuarial senescence or early-life body mass and later-life fecundity. Such phenotypic correlations may lead to differential ageing patterns across traits. Traits may also differ in their sensitivity to environmental factors, and thus in the amount of deterioration accumulated over time, contributing to variation in senescence rates (Moorad & Ravindran, 2022). In addition, traits that are more energetically costly may senesce faster than traits that require fewer energy resources (Bouwhuis et al., 2009; Bouwhuis & Vedder, 2017a). From an evolutionary perspective, natural selection should play an important role in the asynchrony of senescence between traits. Moorad & Ravindran (2022) proposed that the traits that are under the strongest selection early in life senesce the fastest later in life, due to a faster decline of selection with age for these traits. They argue that this pattern may arise from constraints imposed by genetic correlations and outline two potential scenarios. First, traits showing stronger negative genetic correlations between early- and late-life performance should evolve faster senescence than traits with positive or weak among-age correlations. Second, at a given age, negative genetic correlations between fitness traits may reduce the strength of selection acting on each trait, resulting in weaker senescence in negatively correlated fitness traits (and faster senescence in positively correlated traits).

Despite the wide evidence of inter-individual differences in lifespan and senescence patterns, much remains unknown about the factors driving these differences (Gaillard & Lemaître, 2020). Importantly, in the wild, environmental conditions play a significant role in shaping life-history trade-offs and mortality risks while interacting with individuals' intrinsic characteristics to shape ageing trajectories. Building on the theoretical foundations outlined above, which provide an evolutionary perspective on why ageing evolves, this thesis investigates how intrinsic factors (such as sex and genetic variation), extrinsic factors (such as environmental conditions), and transgenerational effects (parental age effects) modulate ageing, thereby providing new insights into the determinants of ageing diversity in the wild.

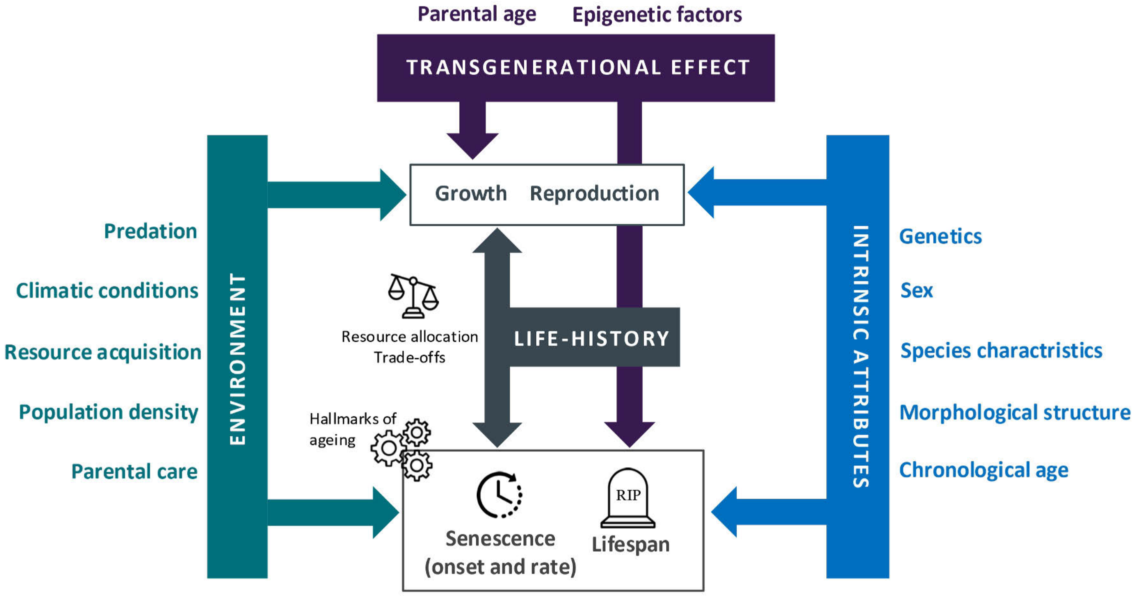

Figure 1. Schematic summary of factors influencing ageing and longevity.

1.5 Intrinsic factors affecting ageing: genetic factors

1.5.1 Genetic factors affecting ageing

Genetic factors can play an important role in inter-individual variation in longevity (Rose, 1991). According to the evolutionary theories of ageing, the existence of genes selected exclusively to induce ageing is unlikely. However, alleles with pleiotropic effects, which have positive effects early in life but deleterious effects emerging late in life, can influence ageing and longevity (Kirkwood & Austad, 2000). Moreover, longevity can be shaped by genes that regulate activities such as DNA repair and oxidative stress management (Kirkwood & Austad, 2000). Research using model organisms such as yeast, *C. elegans*, *Drosophila*, and mice has revealed specific gene mutations (e.g., *age-1*, *daf-2*, *mth*) that are associated with increased longevity (Friedman & Johnson, 1988; Kenyon et al., 1993; Lin et al., 1998; Partridge, 2010). Moreover, empirical studies in human populations have estimated that genetic factors account for approximately 25% of the variability in individual longevity (Herskind et al., 1996; Ljungquist et al., 1998; Skytthe et al., 2003). In the past decade, the development of genome-wide association studies (GWAS), statistical analyses quantifying the association of genetic variants with specific phenotypes (traits or diseases) across the genomes of many individuals, has allowed identifying multiple genes associated with lifespan and age-related diseases (Caruso et al., 2022; Melzer et al., 2020). In particular, the variants of two genes, *APOE* and *FOXO3A*, were shown to be associated with longevity in multiple human populations (Caruso et al., 2022; Melzer et al., 2020). Therefore, it is now well established that ageing and longevity are shaped by multiple genes that drive the inter-individual variability in ageing (Caruso et al., 2022; Kirkwood & Austad, 2000; Melzer et al., 2020; Zhang et al., 2023).

1.5.2 Quantitative genetics

Different approaches have been used to identify genes involved in ageing and to quantify its heritability, such as GWAS, quantitative trait loci (QTL) mapping, transcriptional analyses of age-related changes in gene expression, and selective breeding for slow or fast rates of ageing to identify key genomic regions or candidate genes (Campisi et al., 2019; Melzer et al., 2020; Poirier & Seroude, 2005; Wheeler & Kim, 2011). Despite growing interest in ageing research, the extent to which genetic, environmental, and their interactive effects contribute to variation in longevity remains unclear (Briga & Verhulst, 2015). To explore these influences in the wild, many studies have applied quantitative genetic analyses. This approach estimates heritability and partitions phenotypic variance into genetic and environmental components based on social or genetic pedigree (Charmantier et al., 2014). A key parameter in this framework is the narrow-sense heritability (h^2), defined as the ratio of additive genetic variance (V_A) to the total phenotypic variance (V_P), i.e., $h^2 = V_A/V_P$ (Charmantier et al., 2014). The total phenotypic variance is the sum of the variance due to genetic sources, such as additive, dominance, and epistatic effects (V_G), and environmental variance (V_E), resulting in the expression: $h^2 = V_A/(V_G+V_E)$. Importantly, V_A represents the proportion of phenotypic variation of a trait attributable to additive genetic effects and reflects the evolutionary potential of a trait under selection (de Villemereuil et al., 2019; Forester et al., 2022; Hoffmann et al., 2017).

1.5.3 Fitness and fitness-related traits

In his fundamental Theorem of Natural Selection, Fisher (1930) stated that “the rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time” (Fisher, 1930). This implies that populations with higher genetic variance in fitness can experience more rapid evolutionary improvement. The interpretation emerging from this paradigm was that fitness and traits closely related to fitness, such as reproduction and lifespan, are expected to be under strong directional selection, which will reduce their additive genetic variance (Fisher, 1958; Robertson, 1955, 1966). In addition, when estimating a trait heritability, a low value can be obtained not only from low additive genetic variance (V_A), but also from high environmental variance (V_E). Consequently, fitness and fitness-related traits, especially in natural populations where environmental influences are expected to be strong, are generally predicted to exhibit low heritability (typically ≤ 0.2; Visscher et al., 2008). Initial support for this theory came from studies in domesticated and laboratory animals (Falconer, 1989), and was later extended to wild populations (review by Mousseau and Roff, 1987). Although several studies found low heritability of fitness validating the theory, for example, in bird populations (Brommer et al., 2007; De Villemereuil et al., 2019; Gustafsson, 1986; McCleery et al., 2004; Merilä & Sheldon, 2000; Moiron et al., 2022; Teplitsky et al., 2009; Wheelwright et al., 2014), a recent meta-analysis on long-term data from 19 wild populations of birds and mammals, and found relatively high estimates of V_A for fitness, on average twice as large as previously reported in the literature (Bonnet et al., 2022). The study thus highlighted a stronger-than-expected potential for natural selection to shape fitness and fitness-related traits in wild populations.

1.5.4 Additive genetic variance and heritability of lifespan

By mediating individuals' lifetime reproductive success, lifespan is a key component of fitness (Merilä & Sheldon, 2000). When measured, both traits often appear highly correlated (e.g., Kruuk et al., 2000; McCleery et al., 2004). Therefore, the V_A and h^2 of lifespan are expected to be low (Robertson, 1966), especially in wild populations. However, results across various species do not always support this hypothesis. In human populations, the heritability of lifespan has been estimated to range between 0.20 and 0.30 (Herskind et al., 1996; Ljungquist et al., 1998; McGue et al., 1993; Mitchell et al., 2001; Skytthe et al., 2003), with some estimates reaching as high as 0.50 (Yashin and Iachine, 1995; Yashin et al., 1999). Since the eighteenth century, mortality from infectious diseases and environmental factors has markedly declined in human populations, thanks to improved nutrition, living conditions, and progress in medicine and public health (Cutler et al., 2006). This decline in mortality due to non-genetic factors may explain the high heritability of lifespan measured in human studies. However, recent work found lower estimates ($h^2=0.16$; Kaplanis et al., 2018) and one study demonstrated that previous estimates were inflated due to assortative mating and may be less than 0.10 after correcting for these effects (Ruby et al., 2018). In laboratory and captive animals, a study on killifish (*Nothobranchius furzeri*) found a high estimate of 0.32 (Kirschner et al., 2012), while estimates of 0.44±0.15 and 0.62±0.1 (mean±SE) were found in two mouse populations (Klebanov et al., 2000). Similarly, a comparative study across 22 species found high heritability estimates in several species, such as lions (*Panthera leo*, 0.55±0.14), and ringed teal, (*Callonetta leucophrys*, 0.41±0.24) (Ricklefs & Cadena, 2008). Because laboratory and captive animals are kept in controlled environments with lower extrinsic mortality risks, their heritability of lifespan may be overestimated relative to wild populations (Briga & Verhulst, 2015).

In natural populations, quantitative genetic studies are limited as they require large pedigrees and long-term individual-based monitoring (Moiron et al., 2022; Postma, 2014). Among these studies, some have reported heritability estimates of lifespan close to 0 (e.g., Kruuk et al., 2000; McCleery et al., 2004; Coltman et al., 2005; Schroeder et al., 2012; Vedder et al., 2022). However, some found moderate or even high values, for example, 0.43±0.08 (mean±SE) in semi-free-ranging female macaques (*Macaca mulatta*, Blomquist, 2010), 0.46±0.24 and 0.32±0.42 in two wild populations of Bighorn sheep (*Ovis canadensis*, Réale and Festa-Bianchet, 2000), and 0.21±0.11 in wild male Red-billed gulls *(Larus novaehollandiae scopulinus*, Teplitsky et al., 2009).

Taken together, these findings indicate substantial heterogeneity in lifespan heritability across species and populations. Moreover, studies estimating h^2 and V_A separately in males and females, or assessing how these parameters vary with age, remain rare (Kruuk et al., 2008). Importantly, genes shared between sexes may be expressed in a sex-specific manner or may have opposite effects in males and females. Such sex-specific genetic architecture is quantified by the cross-sex additive genetic correlation (r_A). This correlation can be positive, indicating that genetic factors affect a trait in the same direction in both sexes, null, or negative. The latter indicates sexual conflict, whereby genetic factors affect a trait in opposite directions in males and females, potentially constraining its evolution (Connallon & Matthews, 2019; Kirkpatrick, 2009; Kruuk et al., 2008). The additive genetic variance in males, females, together with the cross-sex genetic correlation, jointly determine the total additive genetic variance of a trait in a population and are therefore critical for understanding evolutionary dynamics (Kirkpatrick, 2009; Kruuk et al., 2008; Shaw & Shaw, 2014; Walling et al., 2014). Consequently, estimating $V_{A,}$ h^2, and cross-sex r_A for lifespan is essential for understanding both the diversity of this trait in a population and its evolutionary potential in response to selection. However, such studies are rare in natural populations, particularly in long-lived species.

1.6 Intrinsic factors affecting ageing: Differences between males and females

1.6.1 Sex-specific ageing

The sex of individuals plays a fundamental role in shaping individual ageing rates and lifespan, contributing to the observed diversity in ageing patterns. Sexes differ in their mortality rates and lifespan, as evidenced in various species (Austad, 2011;

Austad & Fischer, 2016; Liker & Székely, 2005; Marais et al., 2018), although various mammal (Austad & Fischer, 2016) and bird species (Liker & Székely, 2005) show no sex difference. The magnitude of the sex difference in ageing rate and lifespan varies widely among species. In a comparative analysis across 101 mammal species within 134 wild populations, Lemaître et al. (2020) estimated that this variation reaches up to 182% for lifespan and 291% for ageing rate. In mammals, including humans, males typically exhibit higher mortality rates and shorter lifespans than females (Austad, 2006, 2011; Lemaître, Ronget, Tidière, et al., 2020; Nussey et al., 2013; Rochelle et al., 2015; Zarulli et al., 2018). On average, female mammals have a median lifespan that is 18.6% longer than that of males (Lemaître, Ronget, Tidière, et al., 2020). Interestingly, the opposite pattern is often observed in birds, where females generally experience higher mortality rates and shorter lifespans than males (Liker & Székely, 2005; Marais et al., 2018).

In addition to differences in mortality rate and lifespan, males and females also exhibit different onset and rates of senescence in various phenotypic traits. A wide range of studies in birds and mammals have documented sex differences in the senescence of reproductive traits, in both polygynous and monogamous species (Comizzoli & Ottinger, 2021; Nussey et al., 2013). For example, in the wild Soay sheep, females' reproductive output declines with age, whereas it increases before reaching a plateau in males (Hayward et al., 2015). In albatrosses (*Diomedea exulans*) and red deer (*Cervus elaphus*), both sexes exhibit reproductive senescence, but the rate of decline is faster in males than females (Nussey et al., 2009; Pardo et al., 2013). Studies comparing the senescence rates of other phenotypic traits between sexes, such as biometric traits (e.g., body mass; Hämäläinen et al., 2014; Tafani et al., 2013) or parasite burden/immunity (Beirne et al., 2016; Hayward et al., 2015), are particularly rare. More generally, there is a lack of studies investigating senescence in both sexes in the wild and comparing their patterns (Nussey et al., 2013).

1.6.2 Sexual size dimorphism leading to sex-specific mortality

Several hypotheses have been proposed to explain how selection can lead to such sex differences in lifespan and ageing rates. A well-studied factor is the difference in exposure to extrinsic mortality. For the sex with the highest extrinsic mortality, in other words, with the highest environmentally imposed mortality, selection will be weak, leading to shorter lifespan and a faster rate of senescence (Bonduriansky et al., 2008; Gaillard & Lemaître, 2017; Williams, 1957). The sex-specific difference in ageing is expected to be particularly visible in polygynous species that show clear phenotypic differences between males and females. Polygynous males are usually larger, display costly secondary sexual characteristics, and are subject to higher extrinsic mortality due to male-male competition, and are thus expected to have shorter lifespans and faster rates of actuarial senescence (Bronikowski et al., 2022; Clutton-Brock & Isvaran, 2007; Nussey et al., 2013; Tidière et al., 2015). A comparative study on wild mammals confirmed this idea by finding a higher rate of actuarial senescence in males than females, with a difference increasing with the degree of polygyny (Clutton-Brock & Isvaran, 2007). Another comparative study on captive ruminants found shorter lifespans in males than females, with a sex difference clearly higher in polygynous than monogamous species (Tidière et al., 2015). As opposed to mammals, most birds are socially monogamous, as observed in a comparative study across 41 avian families. Males thus experience less male-male competition, which could explain the opposite pattern observed in avian species (Liker & Székely, 2005). Moreover, the few polygynous birds in this study showed male-biased mortality, similarly to mammalian patterns, thus confirming the hypothesis (Liker & Székely, 2005).

Importantly, recent works have re-examined and nuanced previous results linking large sexual size dimorphism, higher extrinsic mortality, with lower lifespan, and accelerated ageing rates (Moorad et al., 2019). Lemaître, Ronget, Tidière, et al. (2020) found that the sex difference in lifespan across mammals was only weakly associated with sexual size dimorphism and found no consistent sex difference in the rates of actuarial senescence. The authors suggest that the sex differences in ageing phenotypes (i.e., lifespan and ageing rates) are shaped by interactions between the cost of secondary sexual traits, environmental factors, and sex-specific susceptibility to adverse conditions (Lemaître, Ronget, Tidière, et al., 2020). The investment of males in the development and maintenance of costly secondary sexual characteristics can make them more vulnerable to adverse conditions. Similarly, a study on the polygynous wild boar (*Sus scrofa*) found that males are larger and display higher mortality than females, but that both sexes have similar rates of actuarial senescence (Gamelon et al., 2014). In addition, it is important to distinguish between lifespan and senescence rates (Monaghan et al., 2008). A recent study across 96 captive mammal species found a low association between lifespan and actuarial senescence (Péron et al., 2019). A similar result was observed in humans: although men have a shorter lifespan than women, the rate of actuarial senescence is similar between the sexes (Austad & Fischer, 2016; Vaupel, 2010).

1.6.3 Sex-specific life-history strategies

The differences in ageing and lifespan between males and females may also involve sex-specific life-history strategies (Bonduriansky et al., 2008; Brooks & Garratt, 2017). The sex investing more in reproduction, parental care, or other energetically demanding activities (e.g., growth, foraging, territorial defense, secondary sexual characteristics) would experience increased rates of senescence (Murgatroyd et al., 2018; Nussey et al., 2009; Reed et al., 2008; Reid et al., 2003). For instance, males may allocate more resources into growth, development, and maintenance of costly secondary sexual characteristics, at the expense of the maintenance of their soma (e.g., cellular repair mechanisms), and may express behaviours exposing them to higher mortality risk compared to females, thus leading to shorter lifespan and faster ageing rates (Bonduriansky et al., 2008; Brooks & Garratt, 2017; Marais et al., 2018). In red deer, higher allocation to early sexual competition in males was shown to be at the expense of faster reproductive senescence in red deer (Lemaître et al., 2014). In barn swallows *(Hirundo rustica)*, males show a cost of developing long tail feathers at a young age, as they have a shorter lifespan (Balbontín et al., 2011). As opposed to polygynous species, we know less about the sex-specific senescence rates and lifespan in monogamous ones (Nussey et al., 2013). These sex differences are less expected than in polygynous species, as males and females show low sexual dimorphism and more balanced investment in reproduction and parental care between sexes (Clutton-Brock & Isvaran, 2007; Promislow, 2003). Nonetheless, differences in ageing can also evolve in monogamous species if both sexes differ in their optimal investment into reproduction and thus in their life-history strategies (Lemaître & Gaillard, 2017; Nussey et al., 2008). Therefore, more studies on monogamous species are needed, especially on wild populations, since most results come from captive organisms (Tidière et al., 2015).

1.6.4 Molecular and physiological mechanisms

At the physiological level, sexual dimorphism is partly explained by hormonal differences between males and females in vertebrates. In particular, the higher levels of androgen hormones in males play a key role in the development of secondary sexual traits such as ornaments and weapons (Brooks & Garratt, 2017; Marais et al., 2018). However, higher circulating levels of androgens, such as testosterone, impair the immune system and reduce immunity, which increases males' vulnerability to pathogens and ultimately their survival (Bonduriansky et al., 2008; Foo et al., 2017; Marais et al., 2018). On the contrary, high levels of oestrogen in females can have protective properties that may increase their survival rate and contribute to the sex difference in ageing and lifespan (Foo et al., 2017). More generally, males appear to have a lower immunity, which is not necessarily a consequence of higher androgen levels. Multiple mechanisms would be at play, as reviewed in (Metcalf et al., 2020).

The mother's curse hypothesis provides another potential explanation for longer female longevity. Because mitochondrial DNA is maternally inherited, natural selection acts only through the female lineage. As a result, mutations that are deleterious primarily in males would not be efficiently removed by selection

(Maklakov & Lummaa, 2013; Marais et al., 2018). These male-biased harmful mutations could therefore persist and accumulate in mitochondrial genomes across generations, potentially increasing mortality and reducing lifespan in males (Frank & Hurst, 1996; Maklakov & Lummaa, 2013; Marais et al., 2018).

At the molecular level, an important sex difference lies in the sex chromosomes, which influence ageing and longevity. Across taxa, the heterogametic sex, males in mammals (XY) and females in birds (ZW), tends to exhibit higher mortality than the homogametic sex (Marais et al., 2018; Trivers, 1985). One key mechanism proposed to explain this pattern is the "unguarded X" hypothesis: in the heterogametic sex, any deleterious mutation on the X or Z chromosome is fully expressed because there is no second homologous chromosome to mask its effects. In contrast, in the homogametic sex, expression of such mutations depends on the individual's zygosity and whether the mutation is recessive or dominant. As a result, the heterogametic sex is more exposed to deleterious alleles, which can lead to shorter lifespan and higher mortality (Marais et al., 2018; Trivers, 1985). Other mechanisms linked to sex chromosomes have also been proposed (reviewed in Marais et al., 2018). For example, the "toxic Y" hypothesis suggests that the Y chromosome harbours a higher density of transposable elements that can increase mutation rates, disrupting genomic integrity, and accelerating male ageing.

1.7 Extrinsic factors affecting ageing

1.7.1 Environmental conditions

In the wild, organisms live in fluctuating environments and are exposed to a variety of stressors and hazards, making the environment a major source of extrinsic mortality. Empirical studies on birds and mammals found higher extrinsic mortality rates in wild populations relative to those kept in captivity (Bronikowski et al., 2002; Ricklefs, 2000). Beyond increasing mortality risk, environmental pressure can have a significant impact on the ageing process of wild animals, shaping individuals' life-history trajectories, senescence patterns, and lifespan. This was demonstrated in a study on stalked-legged flies, which evidenced a striking difference in lifespan and senescence between wild and captive cohorts (Kawasaki et al., 2008). Flies in the wild were exposed to high extrinsic mortality when compared to laboratory flies, and in turn, senesced twice as fast and had a dramatically shorter lifespan than laboratory flies (Kawasaki et al., 2008). In the wild, a longitudinal study on Soay sheep explored the relative influence of the environment and chronological age in shaping individuals' variation in parasite resistance throughout their lives (Hayward et al., 2009). The study showed that the cumulative environmental stress individuals experience over their lives shapes the rate of senescence in parasite resistance. This result highlights the importance of extrinsic factors in shaping ageing trajectories and rates in organisms, thereby contributing to the diversity of ageing patterns observed in nature. Consequently, it is essential to consider environmental factors alongside age-related variation when studying ageing in wild populations.

1.7.2 Physical and social environment

The environment can be divided into two components: the physical environment, which encompasses factors such as climate, food availability, and pathogen exposure; and the social environment, which involves interactions among organisms, including predation, cooperation, and parental care. As mentioned above, the environment is an important source of extrinsic mortality. For instance, exposure to pathogens and adverse climatic events can directly increase mortality and accelerate the ageing process. On the other hand, some environmental factors can lead to the opposite effect. Dietary restriction is a widely studied and good example. This physical factor has an important influence on the ageing phenotype of individuals by shaping their life-history trajectories. There is now numerous experimental evidence that dietary restriction delays ageing, protects against age-related diseases and extends lifespan across distant species (e.g., yeast, nematode worms, flies, fish, rodents, dogs, rhesus monkeys; Fontana et al., 2010; Fontana & Partridge, 2015; T. B. L. Kirkwood & Shanley, 2005).

Regarding the social environment, living in a group presents costs such as competition for access to resources and breeding partners (Gesquiere et al., 2011; Lucas & Keller, 2020; Schoepf & Schradin, 2012; Verhulst et al., 2014), as well as increased exposure and transmission of parasites, infections, and diseases (Altizer et al., 2003; Kappeler et al., 2015; Lucas & Keller, 2020). These factors can negatively affect health, survival, and can accelerate the rate of senescence. In addition, when resources are limited, increasing population density and competition for resources, in association with the ecological context, can influence growth, reproduction, and survival, such as in ungulates (Clutton-Brock, Albon, et al., 1987; Clutton-Brock, Major, et al., 1987; Festa-Bianchet et al., 2003). Also, population density can shape the patterns of senescence. In red deer, for example, increasing population density was evidence to decrease the onset and increase the rate of senescence in body mass (Mysterud et al., 2001). Despite the costs of living in a group, sociality appears beneficial towards ageing and lifespan. A recent study by Salguero-Gómez (2024), examining 152 species, found a relationship between a species' level of sociality and its life-history traits. Specifically, more social species tended to have a slower pace of life, including increased longevity. However, the study found no significant relationship between sociality and the rates of reproductive or actuarial senescence (Salguero-Gómez, 2024). The social environment can influence the survival and longevity of individuals through various processes reviewed in Lucas & Keller (2020). Belonging to a group can reduce extrinsic mortality, a prerequisite for the evolution of longer lifespans, through the reduction of predation risk, cooperation in the acquisition of resources, and cooperation in rearing offspring, especially kin (Bourke, 2007; Lucas & Keller, 2020). In cooperative breeders, more specifically, their longer lifespan and slower ageing rate can result from reduced costs of reproduction with the presence of helpers (Downing et al., 2021; Hammers et al., 2019, 2021), delayed age at first reproduction (Kreider et al., 2022), and higher allocation of resources to self-maintenance thanks to the division of labour (Hammers et al., 2019; Lucas & Keller, 2020).

1.7.3 Importance of the early life environment

Environmental factors affect individuals throughout their lives. The early life period, more specifically, is increasingly recognised as a crucial time in shaping ageing trajectories (Monaghan et al., 2008). There is numerous evidence, including in wild populations, that environmental factors (both physical and social) experienced early in life, before maturity and during foetal development, can have long-term effects on fitness (Berger et al., 2015; M. Douhard et al., 2014; Kruuk et al., 1999; Martínez-Padilla et al., 2017; Vasilieva & Tchabovsky, 2020), life history, and other phenotypic traits of individuals (De Kogel, 1997; Descamps et al., 2008; Hamel et al., 2009; Lindström, 1999; Metcalfe & Monaghan, 2001; Nussey et al., 2007; Post et al., 1997; Reed et al., 2008). More specifically, an adverse early-life environment, such as low-quality parental care, low resource availability, adverse weather conditions, and high population density, can negatively affect adult mortality, accelerate ageing, and reduce lifespan (Monaghan et al., 2008; Monaghan & Haussmann, 2015; Nussey et al., 2013; Vasilieva & Tchabovsky, 2020). A study on three large herbivores population, mountain goats (*Oreamnos americanus*), roe deer (*Capreolus capreolus*) and bighorn sheep (*Ovis canadensis*), found that individual quality, defined by longevity, last breeding success, adult mass and social rank, decreased when individuals were born in years marked by unfavourable environmental conditions, including harsh weather conditions, limited resource availability, and high population density (Hamel et al., 2009). These early-life conditions accounted for 35% to 55% of the variation in individual quality (Hamel et al., 2009). Early environmental conditions also appear to influence the rates of senescence in wild populations. In a meta-analysis on 8 mammal and 6 bird species, Cooper & Kruuk (2018) found that more favourable early-life conditions were associated with a slower rate of reproductive senescence. However, they found no relationship between early-life conditions and the rate of survival senescence.

1.7.4 Underlying mechanisms

Environmental conditions experienced throughout life can modulate an individual's life-history strategies and resource allocation, thereby influencing its longevity and senescence (Garland et al., 2022; Shanley & Kirkwood, 2000). More generally, both the acquisition of resources (e.g., foraging efficiency, competitive capacity) and their allocation (e.g., growth rate versus survival) are tightly constrained by resource availability and environmental conditions, and shape life-history strategies (van Noordwijk & de Jong, 1986). Under adverse environmental conditions, when resources are scarce, individuals may allocate more energy to self-maintenance at the expense of reproduction, enabling them to survive challenging periods. Once environmental conditions are favourable again, organisms could switch back toward higher allocation to reproduction (Shanley & Kirkwood, 2000). When experienced early in life, environmental factors can modulate the trade-off between growth rate and lifespan (Metcalfe & Monaghan, 2003), as evidenced experimentally in three-spined sticklebacks, *Gasterosteus aculeatus* (Lee et al., 2013). By exposing juveniles to warm or cold temperatures to modify their growth rate, the authors showed that accelerated growth reduced median lifespan by 14.5%, whereas a reduced growth rate increased lifespan by 30.6% (Lee et al., 2013). Physiologically, rapid growth is associated with increased oxidative damage, lower resistance to oxidative stress, faster rate of telomere shortening (Alonso-Alvarez et al., 2006; Geiger et al., 2012), and faster rate of body mass senescence (Douhard et al., 2017), which can all lead to increased mortality and shorter lifespan. Early-life environment can therefore influence ageing by mediating life history trade-offs, for instance between early growth and lifespan.

At the molecular, cellular, and systemic levels, ageing is driven by a set of biological mechanisms collectively known as the hallmarks of ageing. First proposed as nine core mechanisms in 2013 (López-Otín et al., 2013), the framework was recently expanded to twelve hallmarks, and includes, for instance, genomic instability, telomere attrition, epigenetic alterations, mitochondrial dysfunction, and cellular senescence (López-Otín et al., 2023). These hallmarks were initially identified and validated in model organisms such as yeast, nematodes, and fruit flies, and have since also been confirmed in mammals. In evolutionary biology, several of these mechanisms have been used to explain how environmental factors influence senescence and lifespan (Monaghan et al., 2008). For instance, environmental stressors were evidenced to decrease telomere length, increase telomere shortening rate (Chatelain et al., 2020), decrease antioxidant defense (Blount et al., 2003), and increase oxidative stress and cellular damage (Isaksson, 2010; Marasco et al., 2017), which are known to be associated with shorter lifespan and increased rate of senescence (Monaghan et al., 2008). In the context of dietary restriction, insulin/insulin-like growth factor signalling (IIS) and mammalian target of rapamycin (mTOR) pathways have been shown to play a central role in influencing health, ageing, and lifespan (Alic & Partridge, 2011; Johnson et al., 2013). More recently, these pathways were evidenced to respond not only to dietary inputs, but also to a wide range of environmental cues reviewed in Regan et al. (2020), further emphasizing the strong influence of extrinsic conditions on ageing and lifespan in nature. Importantly, these pathways are conserved across taxa, indicating shared genetic traits influencing ageing and the evolution of ageing in the tree of life.

Overall, the relationship between the environment and ageing patterns in natural populations is complex. However, given the substantial impact of environmental factors on ageing phenotypes, it is key to include them in the study of ageing patterns and lifespan (Gaillard & Lemaître, 2020; Nussey et al., 2013).

1.8 Transgenerational factors affecting ageing: Parental age effect

1.8.1 Parental age effects

Transgenerational factors, i.e., environmental, epigenetic, and genetic influences transmitted from one generation to the next and affecting the phenotype or performance of the subsequent generation, are receiving increasing attention in research on the determinants of ageing and lifespan. In particular, parental age is a key driver of offspring phenotype, including ageing and lifespan. The effect of parental age on offspring lifespan was first documented by Bell in 1918. Through demographic analyses of detailed genealogical records from an American family, he observed that children born to older parents had shorter lifespans compared to those born to younger parents. This negative effect was later observed in parthenogenic rotifers by Lansing in 1947, who found that an increased maternal age at birth was associated with a decreased lifespan. This negative effect, now known as the Lansing effect, has also been documented in other laboratory animals and shown to affect additional offspring traits, including survival and reproduction (Benton et al., 2008; Fox et al., 2003; Hercus & Hoffmann, 2000; Kern et al., 2001; Monaghan et al., 2020; Priest et al., 2002; Wang & vom Saal, 2000). In a recent meta-analysis across 15 species, including invertebrates (16 studies), birds (1), and mammals (5), maternal age was found to account for 17-22% of the decrease in offspring lifespan, although this negative effect appeared mainly driven by studies in rotifers and insects (Ivimey-Cook et al., 2023).

1.8.2 Studies in natural populations

Parental age effects have been increasingly investigated in natural populations (reviewed in Fay et al., 2016). However, results are contrasting across species. While some studies found a negative effect of parental age at conception on offspring lifespan or survival in birds (Bouwhuis et al., 2015; Fay et al., 2016; Reid et al., 2010) and mammals (Clutton-Brock, Albon, et al., 1987; Descamps et al., 2008; Hoffman et al., 2010; Kroeger et al., 2020; Reichert et al., 2020), other studies found a positive effect of parental age on offspring survival, such as in birds (Bogdanova et al., 2007) and mammals (Hadley et al., 2007; Rödel et al., 2009). Parental age was also evidenced to affect the reproductive success of offspring, either negatively (Bouwhuis et al., 2015; Hadley et al., 2007; Rödel et al., 2009; Schroeder et al., 2015; Torres et al., 2011) or positively (Kroeger et al., 2020; Reichert et al., 2020), depending on the species studied. More generally, according to a meta-analysis, the maternal age effect on juvenile survival is negative in invertebrates and mammals, but positive in birds (Ivimey-Cook & Moorad, 2020). This is possibly due to biparental care, commonly observed in birds but less prevalent in invertebrates and mammals, which could mitigate the effects of maternal age (Ivimey-Cook & Moorad, 2020).

1.8.3 Underlying mechanisms

Although the mechanisms underlying parental age effects remain poorly understood, several potential pathways have been identified. Increasing evidence suggests that, in addition to the soma, the germline deteriorates with age and accumulates damage (Monaghan & Metcalfe, 2019). As a result, germ cells of older parents may carry more mutations, shorter telomeres, altered DNA methylation, and impaired mitochondrial function, all of which can be transmitted to the offspring and affect their ageing process (Monaghan & Metcalfe, 2019). Another possible explanation involves age-related behavioural changes of the parents. Alongside other phenotypic traits, parental care and food provisioning may decline with age, resulting in reduced care quality that can have long-term consequences for the offspring (Beamonte-Barrientos et al., 2010; Fay et al., 2016). Although interest in parental age effects is increasing, most studies are cross-sectional (Monaghan et al., 2020), focus on only a small part of the offspring's lives, and are restricted, especially in mammals, to maternal age effects, given the complexity of assessing male reproductive success (Fay et al., 2016). Hence, studies using longitudinal data from natural populations to investigate the effects of both maternal and paternal age on male and female offspring remain rare (Bouwhuis et al., 2015; Schroeder et al., 2015; Sparks, Hammers, et al., 2022). However, they are key to improving our understanding of the evolution of ageing rates, lifespan, and their diversity across individuals.

1.9 The study of ageing in the wild: knowledge gap

1.9.1 Challenges of studying ageing

Most ageing research has been conducted in the laboratory on short-lived and genetically homogenous model organisms (Kirkwood & Austad, 2000). Laboratory studies offer clear advantages, notably highly controlled environments that allow testing the effects of specific factors or different selection pressures on ageing, and observing their consequences over several generations (Austad & Hoffman, 2018). Despite their technical advantages, laboratory conditions differ markedly from the stochastic conditions experienced by organisms in the wild, with predation, competition, climatic variation, and parasites. Consequently, results obtained under artificial conditions do not always translate to organisms in their natural habitat (Austad & Hoffman, 2018; Van Voorhies et al., 2005). It is therefore crucial to study ageing in wild populations to gain an accurate understanding of the mechanisms driving ageing diversity.

Yet, investigating ageing in natural populations presents substantial challenges. It requires long-term monitoring of a population, with animals individually identified and followed throughout their lives to record survival, reproduction, and other life-history traits. Moreover, individuals need to live long enough to experience senescence (Nussey et al., 2008). As a result, longitudinal studies in wild populations present important temporal, logistical, and economic constraints, making such data particularly challenging to obtain, despite their exceptional value (Clutton-Brock & Sheldon, 2010). Following the realisation of their importance, long-term, individual-based studies of wild populations have increased since 2000 to test evolutionary theories of ageing and to investigate the factors driving the large inter-individual variation in ageing patterns and lifespan (Nussey et al., 2013). This increase, however, was faster than the increase in the number of new species studied to investigate senescence. In addition, investigating transgenerational factors affecting ageing poses even greater challenges, as it requires multigenerational data that capture both environmental variability and individual heterogeneity. Similarly, quantitative genetic analyses necessitate large pedigrees and are particularly data-intensive (Postma, 2014). Therefore, the effects of genetic and transgenerational factors on ageing remain underexplored in natural vertebrate populations, despite being crucial to gain a full understanding of ageing mechanisms and evolution.

1.9.2 Gap in the literature

Although longitudinal studies on wild populations are increasing, they remain particularly scarce for long-lived species. According to the fast-slow continuum of life histories, long-lived species differ markedly from short-lived species in their life-history strategies, constraints, and their sensitivity to environmental factors (Stearns, 1977; Stott et al., 2024). These differences may result in distinct ageing patterns and underlying mechanisms, underscoring the need for more studies on ageing in long-lived natural populations.

In addition, studies of ageing in the wild are mainly restricted to survival and reproductive traits (Bouwhuis & Vedder, 2017b; Lemaître, Ronget, Tidière, et al., 2020; Lemaître & Gaillard, 2017; Nussey et al., 2013) and to female individuals (Fay et al., 2016; Nussey et al., 2013). This bias is partly due to the complexity of assessing male reproductive success in the wild, where uncertainty over paternity often limits data reliability (Bouwhuis et al., 2009, 2010). Therefore, studies that compare senescence patterns across sexes and traits remain scarce (Nussey et al., 2013). This bias in focusing on one or two traits and on one sex only limits our understanding of senescence and its evolution both within and across species (Lemaître & Gaillard, 2017).

Finally, when studying age-related variation in traits at the population level, it is key to account for the heterogeneity in individual quality (Vaupel et al., 1979) by distinguishing the within- and between-individual effects (van de Pol & Verhulst, 2006). The senescence of an organism (i.e., within-individual variation with age) can be masked by virtual variation at the population level (i.e., between-individual effects), driven by the selective disappearance of individuals of certain phenotypes. For instance, individuals of lower quality, characterised by reduced survival and reproductive success, will die earlier compared to higher-quality individuals. As a result, the remaining older individuals in the population will appear to maintain high reproductive success, thereby masking a potential decline at the within-individual level (Nussey et al., 2008; van de Pol & Verhulst, 2006). A similar bias can arise in investigating parental age effects. If low-quality parents that produce short-lived offspring disappear earlier, older parents in the population are likely to be higher-quality individuals producing longer-lived offspring, potentially masking negative effects of parental age on offspring performance, such as the Lansing effect (van de Pol et al., 2006; Vaupel et al., 1979). Evidence for such between-individual effects appears widespread, for example, in avian studies (Bouwhuis & Vedder, 2017b; Vedder & Bouwhuis, 2018), emphasising the importance of accounting for them in longitudinal studies. Nevertheless, studies that controlled for these effects in natural populations remain relatively rare.

1.10 Aims of the thesis

The goal of this thesis is to provide insights into the factors driving diversity in senescence and lifespan in long-lived wild vertebrates and ultimately to expand our understanding of the ageing process in nature. To this end, I investigated the patterns of senescence (onset and rate) and the genetic and transgenerational factors shaping life-history trajectories and lifespan across individuals, using long-term data from two long-lived species: the Asian elephant (*Elephas maximus*) and the Alpine swift (*Tachymarptis melba*). To study these factors, I adopted an integrative framework that considers both sexes, a wide range of traits, environmental influences, and individual heterogeneity. I thus aimed to get a holistic understanding of why some individuals live longer and age faster than others. Through this thesis, I sought to answer three main questions:

What are the patterns of ageing in a long-lived vertebrate, and how do they differ between males and females?

We still know little about whether senescence affects all traits, whether it occurs synchronously across traits, and whether males and females experience senescence in similar ways, although this knowledge is essential to understanding the evolution of senescence in the wild. In this first Chapter, I used longitudinal data from more than 25 years of individual-based monitoring in a population of Alpine swifts. I investigated age-related variation between sexes and among 11 traits, including six biometric traits, four reproductive traits, and one measure of parasite load. Using threshold models, I analysed senescence patterns for each trait and determined the onset and rate of senescence. I then investigated the asynchrony in senescence among traits and between sexes.

Can transgenerational factors cause inter-individual differences in ageing?

To address this question, I investigated the short and long-term effects of parental age at conception on offspring lifespan and life-history trajectories in Alpine swifts. Advanced parental age at conception can have pronounced consequences for offspring reproduction, health, and survival, with potential implications for population dynamics and the evolution of ageing. However, parental age effects have been studied predominantly in laboratory organisms, with few studies conducted in natural populations. Moreover, studies in the wild usually focus on maternal age effects and consider only a short part of the offspring's lifetime. Using a multigenerational demographic dataset from the Alpine swift population, I examined both maternal and paternal age effects on offspring phenotype early in life (body mass, body size, and ectoparasite load) and life-history trajectories at adulthood, including age at first reproduction, lifetime reproductive success, and lifespan.

Is genetics involved in the inter-individual differences in ageing?

Finally, I analysed whether genetic factors contribute to the inter-individual variation in lifespan and survival. Results from quantitative genetic studies on the heritability of lifespan are mixed, and the relative contribution of genetic and environmental factors to variation in lifespan remains poorly understood in wild populations. Using multigenerational demographic data and pedigree information from a semi-captive population of Asian elephants, I investigated the influence of sex- and age-specific genetic effects on variation in lifespan and survival. Applying quantitative genetic models, I estimated the additive genetic variance and narrow-sense heritability (*h2*) of lifespan separately in males and females, as well as the cross-sex genetic correlation for this trait. I then estimated the additive genetic variance and narrow-sense heritability of survival across three age-categories.

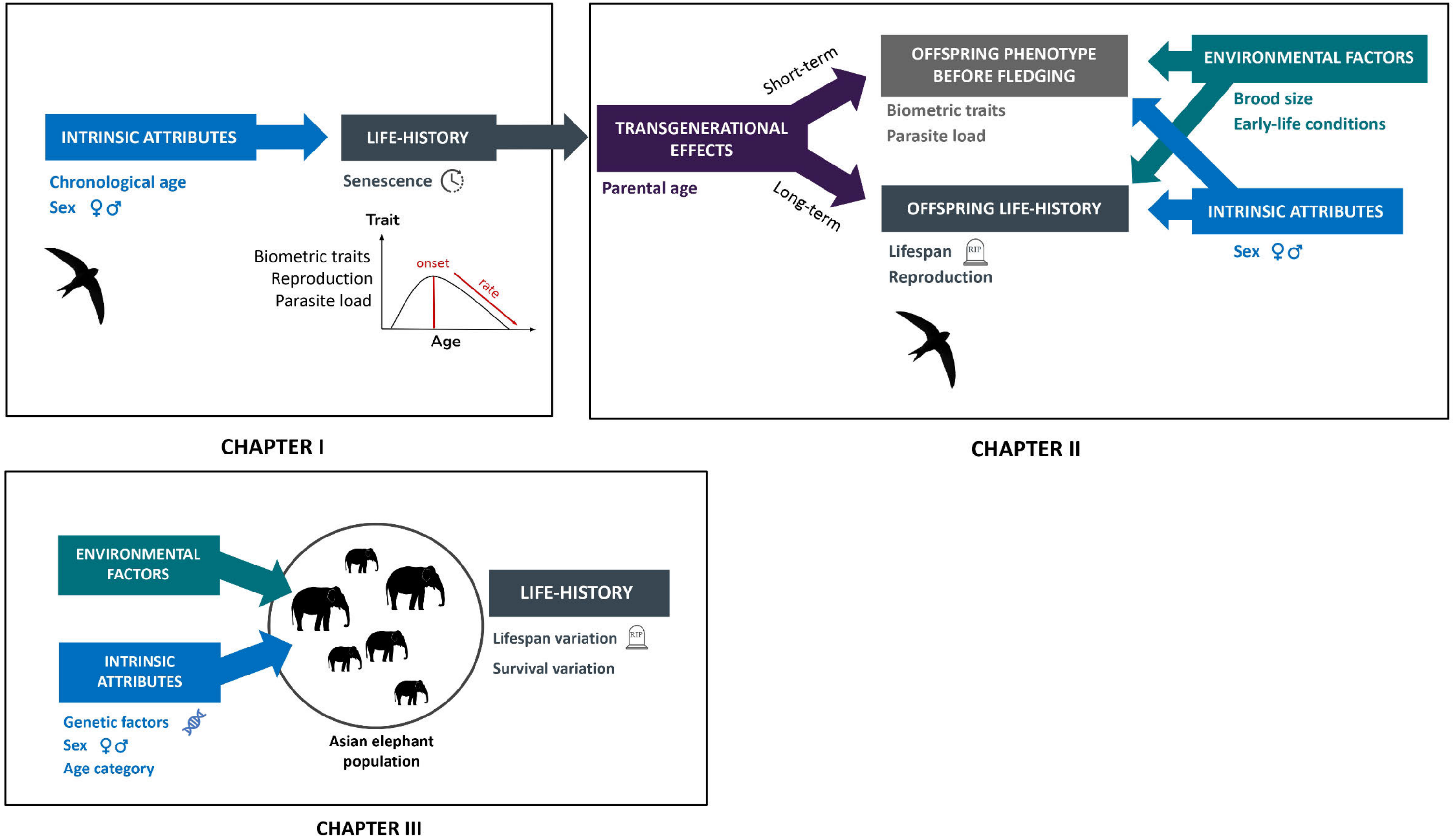

Figure 2. Schematic summary of the aim of the three chapters.

2 Materials and Methods

2.1 Study populations

2.1.1 The Alpine Swift

2.1.1.1 Alpine swift colonies in Switzerland

In **Chapters I** and **II**, I used data on a long-lived bird, the Alpine swift, thanks to a long-term monitoring of four urban colonies located in Switzerland. These colonies are located under the roof of tall buildings in 3 different Swiss cities: Baden (Stadturm: ST, Landvogteischloss: LVS), Biel (Stadtkirche: SK), and Solothurn (Bieltor: BT), have been intensively monitored for 26 years. The database from this monitoring contains detailed demographic data for about 6000 individuals. More than half of the fledglings from this population are recruited locally, natal dispersal is low, and no breeding dispersal is observed (Bize et al., 2017). All adult individuals monitored in the colonies are thus recruits, and a small percentage are young individuals (1 to 2 years) still prospecting before emigrating to an unmonitored colony to start breeding. Every bird is identified with a unique ring attributed as nestlings or when they were first captured in the colonies as adults and had not been ringed before (around 30% of the individuals in the population). This enables the collection of detailed individual data from the nestling stage to adulthood, capturing their full life-history trajectory. Nestlings have been ringed each year since 1932 in Solothurn, 1968 in Biel, and 1991 in Baden, but the data used in this thesis are from intensive individual-based monitoring of adults, including their reproductive success, initiated in 1991 in Baden, 1999 in Solothurn, and 2000 in Biel, and still ongoing to that date. The number of breeding pairs per year for the past 2 decades was 15 in Baden ST, 35 in Baden LVS, 65 in Biel SK, and 50 in Solothurn BT.

2.1.1.2 Alpine swift ecology and life-history characteristics

The Alpine swift is a long-lived bird with a median lifespan of 7 years; the oldest recorded individual was 26 years old (Tettamanti et al., 2012). This bird has a low sexual dimorphism (Dumas, Bize, et al., 2025) and appears sexually monomorphic to human observers. Therefore, individuals are sexed molecularly using DNA extracted from blood or feather samples (Griffiths et al., 1998). The Alpine swift is a migratory and highly aerial bird that spends most of its time flying and lands mainly when breeding. Some individuals have been recorded flying without interruption for over 6 months (Liechti et al., 2013). They breed in colonies of up to several hundred pairs located on cliffs or at the top of tall buildings. Alpine swift reproduction is seasonal. The populations located in Switzerland return from migration in April, and reproduce from May to August, before migrating back in September to the Western part of Africa (Meier et al., 2020). Although the Alpine swift is qualified as a socially monogamous species, a recent study found a rate of divorce of 16.6%, which occurs mainly due to low reproductive success and young age (Dumas, Meier, et al., 2025). Females produce 1 to 4 eggs (on average 3 eggs), which hatch from early May to June, with up to 13 days difference between the first and last egg laid (max 35 days). The eggs then hatch from the beginning of June to early July, and nestlings fledge at around 50 days old (up to 76 days). Both parents incubate the eggs for around 18 days and feed their nestlings until they fledge. Offspring recruit between 1 and 6 years of age, with a mean age at first reproduction of 2.96±0.03 years.

An important factor affecting Alpine swifts' survival is their ectoparasite burden. Alpine swifts are heavily infested by the louse-fly *Crataerina melbae* (Diptera, Hippoboscidae), a hematophagous ectoparasite that impacts the growth (Bize et al., 2003; Bize, Roulin, & Richner, 2004) and survival of nestlings. More specifically, parasitism decreases nestlings' growth rate (Bize et al., 2003), increases their wing feather asymmetry (Bize, Roulin, & Richner, 2004), and increases the mortality of female nestlings (Bize et al., 2005). Louse-flies also affect the reproductive success of Alpine swift (Bize, Roulin, Tella, et al., 2004). The rearing period of experimentally parasitised broods is prolonged by 3 days, which leads to a decline in the reproductive success of parents in the next reproductive event: the number of offspring produced at fledging decreases by 26% compared to the reproductive success of parents from a deparasitised brood (Bize, Roulin, Tella, et al., 2004). To defend themselves from these parasites, Alpine swifts can mount a cutaneous immune response (both adaptive and innate immunity) that affects the blood meal size and survival of the parasites (Bize, Jeanneret, et al., 2008). The cutaneous immune response can impair parasite performance and survival by making the biting site unfavourable for the hematophagous parasites, disrupting their ability to feed efficiently, and causing damage to the parasite tissue (Owen et al., 2010; Wikel, 1996, 1999). In recent years (>2020), Alpine swifts were found to be infected by a trypanosome blood parasite that can lead to acute anemia and death of Alpine swift nestlings (Cigler et al., 2024), with *C. melbae* acting as a competent vector in the transmission of this emerging trypanosomiasis disease (Moré et al., 2025).

The Alpine swifts are insectivorous. They eat insects that they catch in flight, and they drink in flight. It is worth noting that they are, however, unable to eat the louse flies that parasite them. Ectoparasite removal as a defence mechanism appears inefficient and is rarely observed.

2.1.1.3 Data collection

Each year, the different colonies are monitored throughout the whole breeding season, from April to September, by the Swiss Ornithological Institute. Parents are assigned to a nest when captured by hand during incubation or when brooding the nestlings. Nests are regularly controlled to record reproductive parameters from laying date to brood size at fledging, biometric measurements are collected on nestlings and adults, and the ectoparasite load of chicks and adults is reported.

2.1.1.3.1 Reproductive traits

Throughout the breeding season, nests are regularly visited to record laying dates, clutch size, brood size at hatching, and brood size at fledging.

2.1.1.3.2 Biometric traits

Birds' body mass is measured with a digital scale (± 0.1 g). The length of the wing is measured on the flattened and straightened closed wing with a ruler (± 0.1 cm). The length of the tail is measured from the base to the tip of the outermost tail feather with a ruler (± 0.1 cm). The length of the fork is measured as the length difference between the tip of the innermost and outermost tail feathers with a ruler (± 0.1 cm) for both the right and left sides of the fork. The fork fluctuating asymmetry is calculated as the absolute length difference of the two sides of the fork. Because swifts have very short tarsi, the length of the sternum is measured as an alternative to estimate skeleton size. Sternum length is measured with a calliper (± 0.01 cm).

2.1.1.3.3 Parasite burden

Parasite burden is assessed by counting the number of *C. melbae* louse flies in the plumage of the adults and nestlings. *C. melbae* is about the size of a domestic fly, and therefore, this flightless ectoparasite is easy to observe and count.

2.1.1.3.4 Life-history traits

Since each individual is identified with a unique ring and since there is no breeding dispersal in the population, the birds are followed throughout their whole life, and their complete life-histories are known and recorded. More specifically, data on their age at first reproduction, lifespan, and lifetime reproductive success are collected. The lifespan of individuals is assigned when they are found dead or not captured for at least two consecutive years. The lifetime reproductive success is calculated as the total number of fledglings produced throughout the whole life of an individual.

2.1.2 The Asian elephant

2.1.2.1 Myanmar Timber Elephants

In **Chapter III**, I use exceptionally detailed demographic and life-history data from about 9,000 semi-captive Asian elephants. These elephants are owned by the state-owned Myanmar Timber Enterprise (MTE) and work in the timber industry (Schmidt & Mar, 1996). They live in forest camps located in different regions of Myanmar. Although most of the MTE elephants are captive-born, some were captured from the wild before the practice was banned more than 2 decades ago (Lahdenperä et al., 2018; Schmidt & Mar, 1996). They work from 5 to 8 hours a day, 5 days per week, to handle and pull logs from the forest. Pregnant females stop working from mid-pregnancy (11 months into gestation) until their calves reach one year old. Mothers then conduct lighter work. Calves born in captivity are cared for by both their biological mother and other family members, such as grandmothers, aunts, and sisters, called allomothers, which provide care and sometimes allosuckling for the calves (Lahdenperä et al., 2016; Lee, 1987; Lynch et al., 2019). When the calves are approximately 5 years old, they are separated from their mothers to be tamed. The taming process lasts around 4 weeks, after which they return to their natal group or can be relocated to a different group, away from their mothers. Then, they are assigned to a rider called a mahout or oozie, are given an official name, receive a registration number, which is permanently marked on them, and start to be trained (Crawley et al., 2020). From the age of 17–18, the elephants begin working full-time (i.e., 5–8 hours a day). After turning around 55, they retire from work and are cared for in retirement camps. MTE veterinarians carry out monthly check-ups on all the elephants to assess their health, working abilities, and to treat work-related injuries and other illnesses.

Reproduction of MTE elephants occurs without human supervision, with mating and birth happening in the forests, and with no reproductive management of the population. MTE elephants are never culled, and their number is not restricted or regulated. They also forage freely in the forest, and their diet is not supplemented. Therefore, their foraging, breeding patterns, and survival rates are comparable to those of wild Asian elephants (Clubb et al., 2008, 2009; Lahdenperä et al., 2018).

The climate in Myanmar is divided into three distinct seasons: the hot season (February-May), characterised by warm temperatures, monsoon (June-September), which brings heavy rainfall and higher food availability for the elephants, and the cold season (October-January) with milder temperatures. Elephants work during the monsoon and cold season, and rest during the hot season.

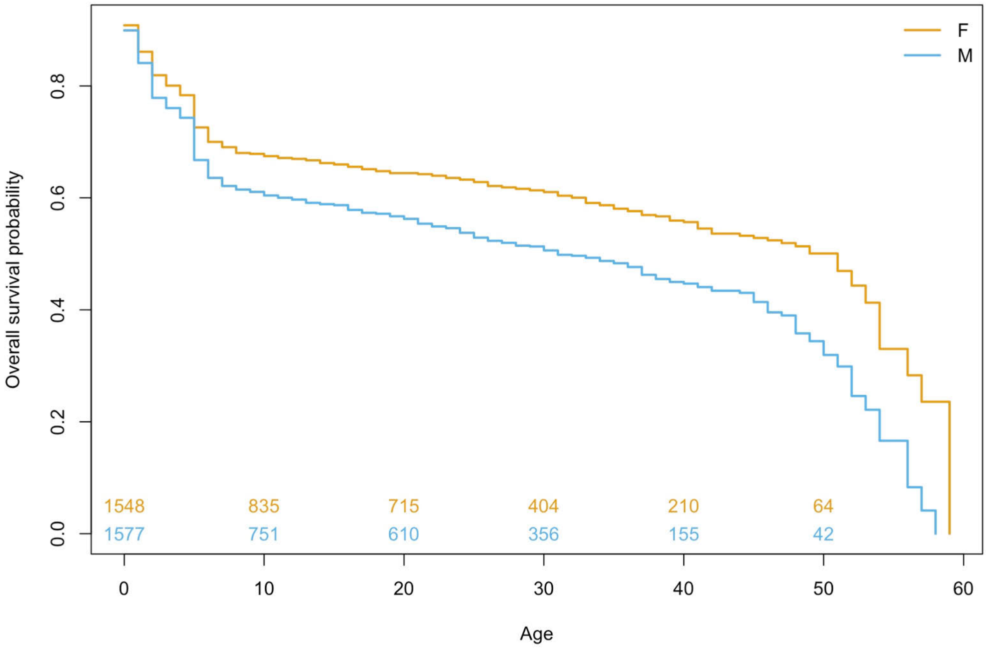

Figure 3. Sex-specific survival curves using a Kaplan-Meier model in MTE elephants between 1960 and 2018. The green and orange survival curves represent males and females, respectively. Figure issued from **Chapter III**.

2.1.2.2 Asian elephant ecology and life-history characteristics

Asian elephants are an extremely long-lived species. MTE elephants have a predicted median lifespan of 30.81 years for males and 44.73 years for females, and they can live over 70 years old (Chapman et al., 2019; Lahdenperä et al., 2018). Elephant mortality is highest during the first year of life, and at all ages, males exhibit higher mortality rates than females (Figure 3; see also Lahdenperä et al., 2018). For both sexes, risk of mortality first decreases with age, with the lowest mortality risk predicted at 13.95 years for females and 14.07 years for males (Lahdenperä et al., 2018), and then increases with age (Figure 3).

Male and female Asian elephants become sexually mature at around 14-16 years, although reproduction by females as young as 8-9 years has been documented, albeit only rarely (Shoshani & Eisenberg, 1982). Asian elephants can reproduce all year round, as female ovulation can occur in any season (Brown et al., 2007; Fowler & Mikota, 2006; Hildebrandt et al., 2011). Females' gestation lasts 24 months, and they give birth to one calf, with a rare occurrence of twins. The interbirth interval is around 4-5 years (Lahdenperä et al., 2014; Sukumar, 2003). Female elephants reach their highest fertility between 20 and 50 years old, with a peak in births occurring around 20-25 years old. Whereas reproductions beyond 60 years have been reported in females (Lahdenperä et al., 2014; Sukumar, 2003), their fertility declines after 50 years (Lahdenperä et al., 2014) and can be followed by a post-reproductive period (Chapman et al., 2019; Lahdenperä et al., 2014). Unlike females, males experience increasing reproductive success with age, resulting in a skew toward older bulls, a pattern well documented especially in African elephants (Moss, 1983; Poole, 1989; Rasmussen, 2005; Sukumar, 2003). Asian elephant bulls continue to grow with age, thereby enhancing their social rank, associated with increasing access to females as observed in both Asian and African elephants (Chapman et al., 2016; Lalande et al., 2022; P. C. Lee & Moss, 1995; Lindeque & Jaarsveld, 1993; Poole, 1989; Sukumar, 2003).

2.1.2.3 Data collection

For over a hundred years, MTE elephants have been monitored by the Myanmar government. Each elephant is marked with a unique identification number on its backside, and a complete set of information, including its life history from birth or capture to death, is recorded and registered in logbooks. The information from these logbooks has later been computerised and saved in a long-term database. This database give access to longitudinal data from each individual such as: their identification number, name, date of birth or date of capture, date of death or last known date alive, origin (captive-born or wild-caught), camp, region, health status, physiological data (i.e., hormones, immunity and other health metrics), faecal parasite counts, morphological data (i.e., height, length, tail length, foot size, chest size, body mass), reproductive events, as well as mothers' identification number and name which give access to a maternal pedigree (genealogy). In this population, the identity of the father is unknown because breeding occurs in the forest, and many individuals are believed to be sired by wild males.

2.2 Statistical analyses

All statistical analyses and graphs in this thesis were produced using R software (R Core Team, 2022), and the figures were built using the ggplot2 package (Wickham, 2016).

In **Chapter I**, I described the ageing trajectories of 11 phenotypic traits in the Alpine swift. The dataset was restricted to individuals of known age (i.e., ringed at nestling or at one year of age, when they still had plumage characteristics distinct from adults) ranging from 1 to 22 years old, that reproduced at least once in their life, and with a complete life-history (i.e., followed from their first reproduction to death). The dataset thus included 244 males and 307 females with 3.8±0.1 observations per individual. I used linear mixed models (LMMs) for each trait and for males and females. For each trait and sex, I compared a series of models: without any effect of age, with age as a linear effect, age as a quadratic effect, and age with different breakpoints known as threshold models (following Berman et al., 2009). I tested models with one threshold varying from 4 to 12 years, and models with double thresholds, one varying from 4 to 12 years and a second from 8 to 16, with at least 3-year intervals in between. To compare the models and determine the one best describing the variation of the phenotypic trait with age, I used the Akaike Information Criterion (AIC). I selected, for each trait and sex, the models with ΔAIC<2 (Burnham & Anderson, 2002). When several models were selected, I used a model averaging approach to obtain the coefficient estimates of all the co-variables in the models. In all mixed models, to tease apart within-individual age effects (experience, senescence) from demographic effects (selective appearance and disappearance), I included as fixed effects the age at first reproduction (AFR) and the lifespan of individuals, following *equation (1)* in van de Pol and Verhulst (2006):

$$r_{ij} = \beta_0 + \beta_W \times age_{ij} + \beta_1 \times AFR_i + \beta_2 \times Lifespan_i + u_{0i} + e_{0ij}$$

A two-level random intercept model with individual i, measurement j, random intercept term u_{0i}, residual error term e_{0ij}, within-individual change ($\beta_W \times age_{ij}$), selective appearance effect ($\beta_1 \times AFR_i$), and selective disappearance ($\beta_2 \times Lifespan_i$). All models also included as fixed factors the colony (Biel-SK, Solothurn-BT), and as random factors the individual identity (ring ID) and year of measurement. I also tested for terminal effects (terminal investment or terminal illness), which can mask the effect of senescence (Bouwhuis et al., 2009; Froy et al., 2013) by including a binomial variable to distinguish the last breeding observation (last observation=1) from all the previous observations (last observation=0).

Following model selection, I calculated from the best models for each trait and sex (i.e., those with ΔAIC<2), the mean age of the second thresholds as the potential age at onset of senescence. Then, for the traits that showed significant variation with age based on the previous models, I conducted additional analyses to determine if, late in life, the trait declines (i.e., senesces), at which rate, and whether there is a difference between sexes. For these analyses, I restricted the data for each trait to late life (i.e., after the second threshold of age for males and females). To increase sample size, I pulled data from both sexes and included individuals with known age and lifespan, but for which we did not know the AFR, thus individuals that started reproducing before the start of the individual monitoring in 2000. To determine the rates of variation, I used LMMs for each model with the individual ID (ring) and year of measurement as random factors, and as fixed effects, the age, sex, lifespan of the individuals, the interaction between age and sex, and the variables that were significant according to the model averaging in the previous approach. All the traits were scaled to compare their rates of late-life variation.

In **Chapter II**, I investigated the effects of parental age at conception on the phenotype at 50 days old and the life history trajectory at adulthood of the offspring in the Alpine swift. I studied the effects of parental age using linear or generalized linear mixed-effect models with the age of the mother and father divided into two age categories, namely “prime-age” versus “old-age” parents. The age threshold between the two age categories corresponds to the earliest onset of senescence determined in **Chapter I**, that is, 11 years of age for the mothers and 9 years of age for the fathers. The analyses were restricted to individuals who were born before 2015 to avoid selective effects towards individuals with a low AFR and a short lifespan. In all models, I controlled for selective disappearance effects by including the lifespan of the mother and father (Berman et al., 2009; Froy et al., 2013; van de Pol & Verhulst, 2006). As early-life conditions can have long-term consequences on individuals’ phenotype (Hamel et al., 2009; Lindström, 1999), I also controlled for the effect of early-life environmental harshness. To do so, I included in the offspring phenotype models the brood size, and in all models the annual mean nestling survival: the average nestling survival until fledging in all colonies at the birth year of a focal offspring, which varied from 55% in 2007 to 96% in 2011. Therefore, to test the parental age effects on offspring life-history trajectories (age at first reproduction, lifespan, lifetime reproductive success), I included as fixed factors the mother and father age category, the lifespan of the mother and father, the sex of the offspring, the annual mean nestling survival, and as random factors the year of birth of the offspring, the colony (Baden-ST, Baden-LVS, Biel-SK, Solothurn-BT) and the parent identity (ring ID). To test the parental age effects on offspring phenotype at 50 days old (biometric traits and parasite load), I included in addition the exact age of the offspring (as it varied slightly from 50 days old for some individuals), the brood size at 50 days, and the hatching date (continuous variable). To analyse parental age effect on LRS and ectoparasite load, I used GLMMs with a Poisson distribution. For all other traits, I use LMMs with a log transformation for offspring lifespan and AFR. In each model, I tested the interactions between parents' age categories and offspring sex, which I removed from the final models if not significant. If significant, I reported the results using the package *emmeans* (Lenth et al., 2023).

In Chapter **III**, I investigated the sex-combined and sex-specific heritability of lifespan, and the age-specific heritability of survival in the MTE Asian elephant population. I used demographic individual-based information on lifespan and survival from captive-born males and females, for which we know the identity of the mother. The dataset included information on elephants until 2022. I restricted the analyses to individuals born between 1960 and 2002 due to the uncertainty of birth dates before 1960 (Jackson et al., 2019) and to avoid a cohort effect with short-lived individuals only represented in the last cohorts. This subset kept a minimum of 20 years of observations. The dataset thus included a total of 3125 individuals (1577 males and 1548 females), with 1275 mothers, of which 2590 had at least one sibling, and with a maximum pedigree depth of three generations. From the 3125 individuals in the dataset, 1260 were reported dead, and 1865 were censored, meaning we do not have their exact death date.

For the analyses, I used the animal model with a Bayesian approach to estimate the additive genetic variance and narrow-sense heritability (h^2) of lifespan, sex-combined and sex-specific, and survival age-specific. I computed the animal model with mixed-effects generalised linear models fitted by Markov chain Monte Carlo (MCMCglmm R package, Hadfield, 2010). For the sex-combined and sex-specific analyses, I included individuals' longevity measured in years as a response variable with a Poisson distribution. For the age-specific model, I included individuals' survival as a response variable, constructed as a binomial variable (0=alive, 1=dead) separated into three age categories: 0-5, 6-19, 20+. For example, an individual who died at 15 years old, as a survival value of 1 in the age category 0-5 and 0 in the age category 6-19. The age categories were chosen to fit with the life-history characteristics and survival curves of MTE elephants, and are consistent with the four age classes regularly used in studies on this population (Reichert et al., 2022; Ukonaho et al., 2023). I constructed a binomial censor variable to distinguish between individuals with a known death date (censor=0) and individuals still alive or lost (censor=1). All models included as fixed effects the sex of the individual, the censor variable (0=still alive or lost, 1=dead), and as random terms the identity of individuals, identity of the mother, birth cohort (per 5 years), and region (11 regions in Myanmar) to control for maternal effects, and temporal and geographical environmental conditions. Hence, the calculation of the heritability of lifespan is as follows:

$$h^2 = \frac{V_A}{V_P} = \frac{V_A}{V_A + V_M + V_{cohort} + V_{region} + V_R}$$

Where the phenotypic variance V_P is the sum of additive genetic (V_A), maternal (V_M), cohort (V_{cohort}), region (V_{region}), and residual (V_R) variances. The variance estimates used to calculate h^2 were extracted from the model's results, following Hadfield (2010). Finally, with the sex-specific model, I also calculated the cross-sex genetic correlation (r_A), using the genetic covariance between sexes:

$$r_A = cov(A\ female, A\ male)/\sqrt{V_{A\ female} \times V_{A\ male}}$$

Where $cov(A\ female, A\ male)$ is the covariance for lifespan between sexes, and $V_{A\ female}$ and $V_{A\ male}$ are the additive genetic variances of lifespan for females and males.

3 Results

3.1 Ageing patterns, onset, and rate of senescence

In **Chapter I**, I found that all the traits studied varied with age except the fork fluctuating asymmetry, length of the sternum, and body mass. In both sexes, the variation with age of the wing length, tail length, parasite burden, laying date, clutch size, brood size at hatching, and fledging was best described with 2-threshold models. For all these traits, we observed a strong improvement early in life (e.g., an increase in tail length, decrease in laying date, and parasite burden), followed by a plateau after the first threshold. The variation observed after the second threshold appeared to decline for some traits (e.g., decrease in tail length and brood size at hatching) but seemed rather stable in a continuity of the plateau for some other traits (e.g., wing length, laying date). Therefore, by pooling data from both sexes to increase the late-life sample size, I then determined the direction and rate of variation after the second threshold to assess if there is a significant senescence of the trait later in life, or if it reaches a plateau. I found a sex-specific decline later in life for the length of the wing, length of the tail, brood size at hatching, and brood size at fledging (Figure 4). The length of the tail declined significantly more rapidly with age in females than in males, and the brood size at hatching and fledging declined significantly in females but not in males, for which it tended to increase (Figure 4). For these senescent traits, I also determined the age at the onset of senescence and found an asynchrony of senescence between traits and sexes. The onset of senescence was significantly earlier in males for tail length, at 9.5 ± 0.6 years old, compared to females, at 15.2 ± 0.4 years old. When comparing the traits within females, which showed senescence in both their biometric and reproductive traits, I observed an earlier senescence for brood size at hatching and fledging (12 ± 0.9 years old) than for tail length (15.2 ± 0.4 years old). Moreover, tail length tended to senescence at a faster rate than brood size at hatching and fledging in females (Figure 4). In both sexes, I also found an effect of individuals' age at first reproduction, lifespan, and last year before death on various traits. Males with a longer and more symmetrical fork had an earlier age at first reproduction, and females that reproduced earlier had higher reproductive performance, as shown by greater brood size at hatching and fledging. Males with a shorter tail and females with higher body mass had a longer lifespan. Finally, in the year preceding death, males produced more offspring at hatching but showed greater fork asymmetry and tended to have lower body mass.

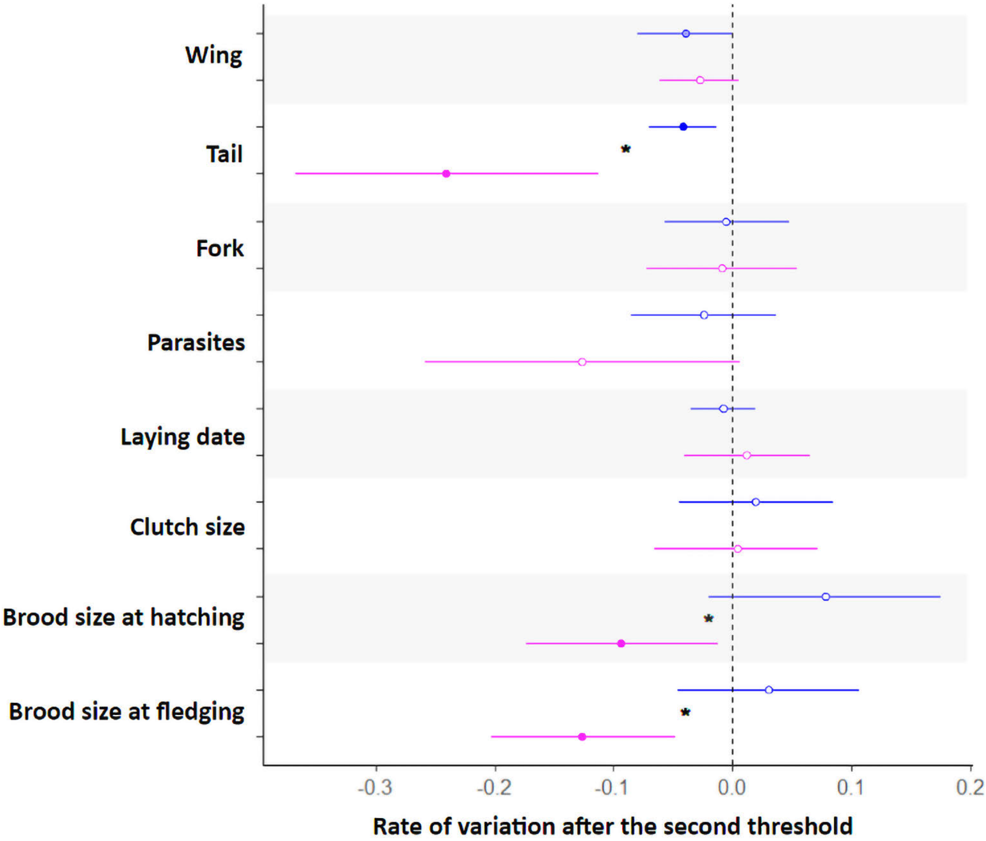

Figure 4. Estimates of ageing rates with 95% confidence in female (pink) and male (blue) Alpine swifts. The analyses for this graph were run on a "late" dataset with pooled observations from both sexes after the onset of senescence (second threshold). The significance of each estimate is indicated by a filled circle when significant ($P < 0.05$), by lightly coloured circles when marginally significant ($0.05 \leq P \leq 0.06$), and by open circles when non-significant ($P > 0.06$). Significant differences in the rate of ageing between males and females for a given trait are indicated by a star symbol. The traits with no evidence of age-related variation (i.e., sternum, fork fluctuating asymmetry, body mass) were removed from these analyses. Figure issued from Moullec et al. 2023, **Chapter I**.

3.2 Parental age effect on offspring traits

In **Chapter II**, the age of the mother did not affect offspring lifespan, but the age of the father had a significant effect on their sons' lifespan, but not on their daughters' lifespan (Figure 5). Sons from old-age fathers were shorter-lived than sons from younger fathers. Sons from older fathers also tended to have a lower LRS (Figure 5, see also sensitivity analyses in supplementary material of Moullec et al., 2025, **Chapter II**), but not daughters. However, parental age at conception had no effect on offspring age at first reproduction (AFR).

When investigating offspring phenotype at fledging, I found no effect of parental age at conception on offspring body mass. However, parental age had a significant effect on offspring body size and parasite burden. Older parents produced bigger offspring with longer wings and tails. Offspring from older mothers tended to have fewer parasites in their plumage. In addition, daughters from older fathers had significantly fewer parasites. However, no significant difference in parasite load was detected between sons from old-age fathers and sons from prime-age fathers. In all models, the lifespan of the parents had no effect, indicating no selective disappearance of lower-quality parents.

In all models (i.e., life-history models and offspring phenotype at fledging models), I also tested the effect of early-life environmental conditions on offspring traits. This early environment was measured as the mean nestling survival from hatching to fledging in all colonies in the year of birth of a focal offspring, and as the brood size at 50 days (for offspring phenotype at fledging models only). I observed a significant positive effect of the annual nestling survival on offspring size, but a tendency for a negative effect on lifespan. Offspring born during harsher years, characterised by a high mortality of nestlings, were smaller at fledging (shorter wings, tail, and sternum), but tended to live longer. In addition, offspring born in bigger broods, thus born in a harsher environment with lower food per capita, were smaller at fledging (shorter wings, tail, and sternum) and had a lower mass.

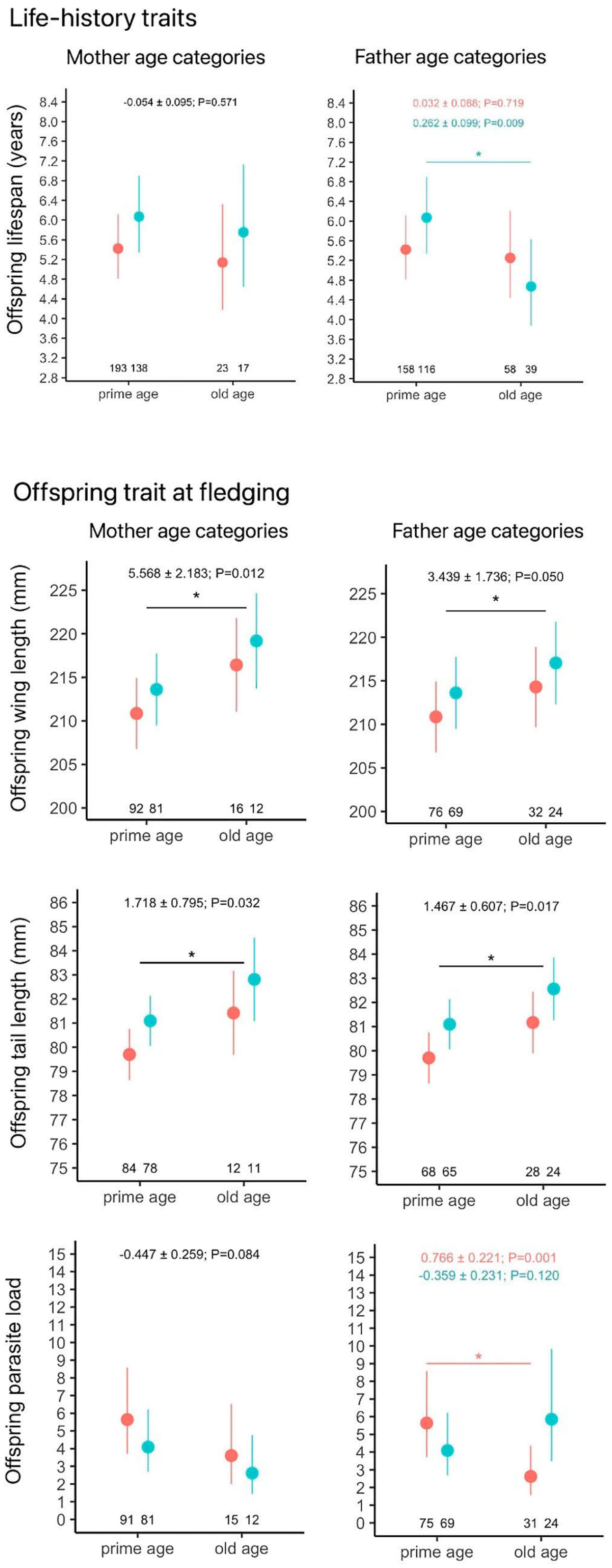

Figure 5. Effects of maternal and paternal age at conception on life-history traits and phenotype at fledging of sons (blue closed points) and daughters (red closed points) in the Alpine swift. The figure only shows the traits for which parental age has a significant effect, i.e., offspring lifespan and wing length, tail length, and parasite load at fledging. The dots with error bars indicate the means ± SE of the predictions of the statistical models. Parents are divided into prime-age (2 to 10 years of age for the mothers and 2 to 8 for the fathers) or old-age (>10 years for the mothers and >8 years for the fathers). Horizontal segments above the panels with an asterisk indicate a significant effect of parental age on both sexes (black), or on sons (blue) or daughters (red) when the interaction between parental age category and offspring sex was significant. The estimate ± standard error and P-value of the parental age effects are indicated above each graph. Sample sizes are reported at the bottom of the panels. Figure issued and adapted from Moullec et al. (2025), **Chapter II,** with only the main effects represented.

3.3 Additive genetic variance of lifespan

In **Chapter III**, I investigated whether genetic factors could explain the variation of lifespan and survival in the semi-captive population of Asian elephants. From the subset of individuals used for the analyses, born between 1960 and 2002 and monitored until 2022, the lifespan varied from 0 to 58 years for males and 59 years for females. Males had a mean lifespan of 15.2 (±16.1 SD) years, as opposed to 17.7 (±16.8 SD) years for females. This lifespan difference was significant, as shown by the posterior mean of the fixed term sex in the sex-combined (-0.137, 95%CI [-0.257; -0.015]) and sex-specific models (-0.179, 95%CI [-0.313; -0.047]).

I found a heritability of lifespan and additive genetic variance close to 0 in the sex-combined model (V_A=0.295, 95%CI [<0.001; 0.635]; h^2=0.057, 95%CI [<0.001; 0.135]; Figure 6). However, I found substantial heritability and additive genetic variance in the sex-specific model for both males (V_A males =1.144, 95%CI [0.328; 2.007]) and females (V_A females=0.428; 95%CI [0.154; 0.743]) (Figure 6), with the 95%CI of V_A shifting away from 0 for both sexes. This substantial V_A led to a nonzero heritability of lifespan for both males (h^2 males=0.209, 95%CI [0.033; 0.406]) and females (h^2 females=0.087, 95%CI [0.015; 0.171]). In addition, I found a low cross-sex genetic correlation, close to 0 (r_A=0.264, 95%CI [-0.236; 0.726]).

Regarding the age-specific model of survival, the additive genetic variance was substantial at the age category 0-5 years (V_A=1.585, 95%CI [0.369; 3.057]), with the 95%CI shifting away from 0, but close to 0 for the age categories 6-19 and 20+. The substantial V_A for the age category 0-5 years led to a relatively high heritability value (h^2=0.326, 95%CI [0.149; 0.509]) with a 95%CI that did not include 0, whereas h^2 was close to 0 for the two older age categories (6-19 and 20+).

In all the models, the geographical region (regions of Myanmar where the timber camps are located), the birth cohort of the elephants (5 years birth cohort), as well as the residual variance for the sex-combined and sex-specific models, explained a large proportion of the total variance, thus showing an important effect of the environment on lifespan and survival variation in this population. On the contrary, the maternal effects variance was close to 0 in all models.

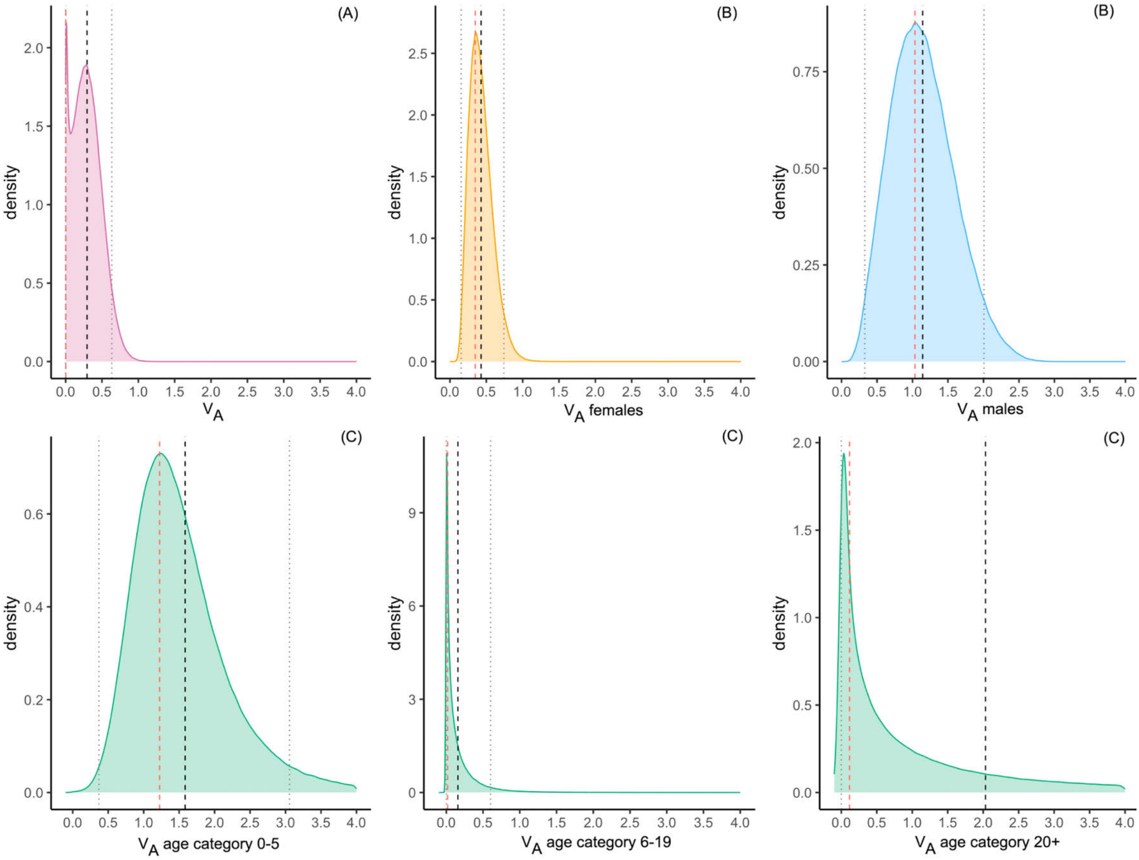

Figure 6. Posterior distribution of the additive genetic variance of lifespan from (A) the sex-combined model, (B) the sex-specific model for females (orange) and males (blue), and (C) posterior distribution of survival from the age-specific model, for the age categories 0-5, 6-19, and 20+ years. Black dashed line: posterior mean. Red dashed line: posterior mode. Grey dotted lines: 95% CI. V_A: additive genetic variance. Note: for the age category 20+, the upper limit of 95% CI was above 8 and therefore does not appear on the graph. Figure issued and adapted from **Chapter III**.

4 Discussion

Understanding why the pace of ageing varies both within and among species is a central question in evolutionary biology. Although senescence is now recognised as a widespread biological process, its expression in natural populations is shaped by a complex interplay of factors that remains to be studied in detail. Using exceptionally rich long-term data from two populations of long-lived vertebrates, the Alpine swift and the Asian elephant, I investigated the factors affecting ageing across individuals' lives and across generations. Through the three chapters of this thesis, I have evidenced intrinsic, extrinsic, and transgenerational factors affecting senescence and lifespan and contributing to ageing diversity in these natural populations. In the following section, I discuss these factors and the potential mechanisms through which they affect the ageing process.

4.1 Senescence affects multiple traits

4.1.1 Most traits vary with age

In **Chapter I**, I modelled the ageing trajectories of biometric traits, reproductive traits, and parasite load in male and female Alpine swifts. Ageing trajectories in the Asian elephant have been previously addressed (Lahdenperä et al., 2014, 2014; Lalande et al., 2022; Lynsdale et al., 2017; Mumby et al., 2013b; Reichert et al., 2022; Robinson et al., 2012). In this thesis, I thus focused on describing ageing trajectories in the Alpine swift, as there was little prior knowledge on trait-specific variation in ageing in this wild long-lived species. Most traits varied significantly with age, except for fork fluctuating asymmetry, body mass, and sternum length. The absence of age-related change in sternum length was expected, as skeletal structures in birds remain fixed once adult size is reached, that is, before fledging in the Alpine swift (Bize et al., 2006). As fork fluctuating asymmetry and body mass showed no significant variation with age, this may reflect overall stability in body condition across the lifespan. Nonetheless, body mass tended to decline slightly with age (see Figure 1 from Moullec et al., 2023, **Chapter I**). In the Alpine swift, body mass shows large daily variations due to environmental factors, such as rainfall and ambient temperature. In another study on the same population that accounted for these daily variations, senescence in body mass was detected in both sexes (Dumas, St. Lawrence, et al., 2024). The remaining traits exhibited clear age-related patterns with two inflection points, typically following a bell-shaped curve consistent with observations in other species (Catry et al., 2006; Frankish et al., 2020; Lecomte et al., 2010; Saraux & Chiaradia, 2022). They showed a marked improvement until 4 to 12 years of age, followed by a plateau and a subsequent decline in later life. In line with the literature documenting ageing processes in wild populations (Gaillard & Lemaître, 2020; Lemaître, Ronget, & Gaillard, 2020; Lemaître & Gaillard, 2017; Nussey et al., 2013), these results provide clear evidence of senescence in this species.

4.1.2 Evidence of senescence in biometric traits

When examining variation with age after the plateau, wing and tail length showed a decline, starting from 9 to 15 years, as similarly observed in the barn swallow (*Hirundo rustica*, Møller and de Lope, 1999). Since Alpine swifts moult annually (Brighten et al., 2025), this reduction may reflect an age-related decline in feather growth, as evidenced for central tail feathers in barn swallows (Adamkova et al., 2022b). For the Alpine swift, a highly aerial bird, senescence in these flight-related traits could have important consequences for flight performance and energy expenditure during flight (Evans & Thomas, 1992; Norberg, 1995a, 1995b; Thomas, 1993), potentially reducing migratory efficiency in older individuals, such as shorter migration distances or delayed arrival at breeding sites, as observed in barn swallows (Møller and de Lope, 1999). Moreover, a decline in wing and tail length could decrease the foraging success of older individuals, as documented in the grey-headed albatross (*Thalassarche chrysostoma*, Catry et al., 2006). Alpine swifts capture insects in flight to feed, for which they have specific long, narrow wings and forked tails, that enable sharp aerial manoeuvres (Norberg, 1995b). Therefore, age-related reductions in these traits could diminish their ability to forage effectively.

4.1.3 Evidence of sex-specific reproductive senescence

Regarding reproductive traits, both males and females showed marked improvement early in life, followed by a plateau, a pattern commonly observed in wild populations (Cooper et al., 2021; Lemaître & Gaillard, 2017). This pronounced early-life increase likely reflects individuals gaining breeding experience (Forslund & Pärt, 1995a; Saraux & Chiaradia, 2022). Since males do not lay eggs, the age-related variation observed in laying date and clutch size likely reflects female mate choice and differential reproductive investment based on mate quality and experience (Komers

& Dhindsa, 1989; Michl et al., 2005; Segami et al., 2021). For instance, females may invest less in current reproduction by delaying breeding or producing fewer eggs when paired with lower-quality or inexperienced males. After the initial improvement and plateau, reproductive senescence was evidenced in females only, marked by a sharp decline in brood size at hatching and fledging beginning around 12 years of age (Figure 4). In contrast, males tended to show increasing breeding performance with age, with a significant increase during their final reproductive attempt. This contrasting trajectory between males and females is consistent with findings in wild Soay sheep, where reproductive senescence has been documented in females but not in males (Hayward et al., 2015). However, it contrasts with patterns of another long-lived, monogamous bird, the wandering albatross, in which breeding probability declines more rapidly in males than females (Pardo et al., 2013). In female Alpine swifts, senescence was evidenced in brood size at hatching and fledging, but not in laying date and clutch size. This suggests that, as similarly observed in great tits (*Parus major*) (Bouwhuis et al., 2009), reproductive senescence is mainly driven by increased egg failure and nestling mortality rather than reduced clutch size. Such declines may result from decreased maternal care efficiency, such as reduced incubation performance, or from age-related deterioration in egg quality and composition (Bouwhuis et al., 2009; Lemaître & Gaillard, 2017).

4.1.4 No evidence of immunosenescence

In many species, the immune capacity of individuals, such as the resistance against parasites, declines with age, a process known as immunosenescence (Hayward et al., 2015; Møller & DE Lope, 1999; Peters et al., 2019). In the Alpine swifts, individuals can defend themselves against ectoparasitic louse flies by mounting a cutaneous immune response (Bize, Jeanneret, et al., 2008). Interestingly, ectoparasite load decreased with age early in life and did not show a significant increase later in life, suggesting no clear evidence of immunosenescence in this species. Similarly, no signs of immune deterioration with age were reported in the Common tern (*Sterna hirundo*), where innate immune parameters remained stable (Bichet et al., 2022), or in the Seychelle warbler (*Acrocephalus sechellensis*), where blood parasite infections did not increase in older birds (Hammers et al., 2016). This result suggests that the senescence of biometric and reproductive traits observed in the Alpine swift is unlikely to be driven by a decline in the cutaneous immune response to ectoparasites. However, this response represents only one component of immune function, and other aspects of immunity may nonetheless deteriorate with age. Immunosenescence could therefore still be present but manifest on different immune pathways. To fully understand how immune function changes with age across the lifespan, direct longitudinal measurements of multiple immune traits are needed in this species, as these would also provide a more comprehensive assessment of individual somatic condition.

4.1.5 Evidence of asynchrony of senescence

By investigating the variation with age in a variety of traits and in both sexes, I found evidence for asynchrony in the onset and rate of senescence both within and among individuals, consistent with the growing evidence in the literature on natural populations (Bouwhuis et al., 2012; Bouwhuis & Vedder, 2017b; Cooper et al., 2021; Fay et al., 2021; Hayward et al., 2015; Nussey et al., 2013). Among individuals, I detected clear sex differences (see section *4.3 Differences between males and females*). Females showed reproductive senescence, whereas males did not, instead showing an increase in brood size at fledging during their final reproductive event. For biometric traits, females displayed a later but steeper decline in tail length compared to males. Within individuals, an asynchrony between traits was observed. In females, the senescence rate of tail length was stronger than that of wing length (Figure 4). For reproductive traits, senescence rate intensified with increasing allocation to reproduction: stronger for brood size at hatching and fledging than for clutch size or laying date, as similarly observed in great tits (Bouwhuis et al., 2009). These differences in the strength and onset of senescence can arise through different mechanisms. For reproductive traits, the differences may reflect the cumulative costs of reproduction, from laying eggs to rearing nestlings until fledging. In addition, the strength of natural selection may vary among traits. Among biometric traits, wing length and shape are more critical for flight aerodynamics and performance than the tail (Thomas, 1997). Additionally, wings show lower asymmetry compared to the tail in forked tail birds (Thomas, 1997). Therefore, natural selection may act differently on these traits (Thomas, 1997), potentially stronger on the wings than the tail to maintain their structure with age. This could account for the steeper senescence in tail length observed in the Alpine swift. However, a recent study from Moorad & Ravindran, (2022) suggests that traits experiencing stronger selection at early ages senesce more rapidly later on because the strength of selection declines faster for these traits. This would suggest, on the contrary, a stronger selection for tail length earlier in life that would result in the observed faster senescence later in life. Understanding the asynchrony in the onset and rate of senescence is important because it can shed light on the mechanisms underlying senescence. For instance, a decline in biometric traits, and thus flight performance, could reduce foraging efficiency and, in turn, lead to a decline in reproductive success. However, although both sexes showed senescence in biometric traits, reproductive senescence was detected only in females. Moreover, when comparing the onsets of senescence, reproductive senescence in females occurred before the senescence of tail length (see Figure A3 in the supplementary material of Moullec et al., 2023, **Chapter I**). Overall, our results do not provide clear evidence that the decline in one trait potentially triggers or influences senescence in another.

4.1.6 Evidence of between-individual effects

For all the traits studied, the increase or improvement early in life was not driven by the selective appearance of "higher quality" phenotypes in the population. Indeed, the age at first reproduction, controlling for selective appearance in the models, was not significant, except for the fork length and fork fluctuating asymmetry in males, and reproductive success in females. Males with longer and symmetrical forks, and females with higher reproductive performance (higher brood size at hatching and fledging), started reproducing earlier. This pattern reinforces the strong within-individual increases observed in both biometric and reproductive traits with age early in life. At older ages, a selective disappearance effect on males' tail length was detected: males with longer tails had shorter lifespans, which may have partly contributed to the observed decline in tail length. In addition, females with higher body mass tended to have a longer lifespan, suggesting that higher-quality females tend to live longer (Tettamanti et al., 2012; Weladji et al., 2006), which on the contrary may have masked a potential decline in body mass.

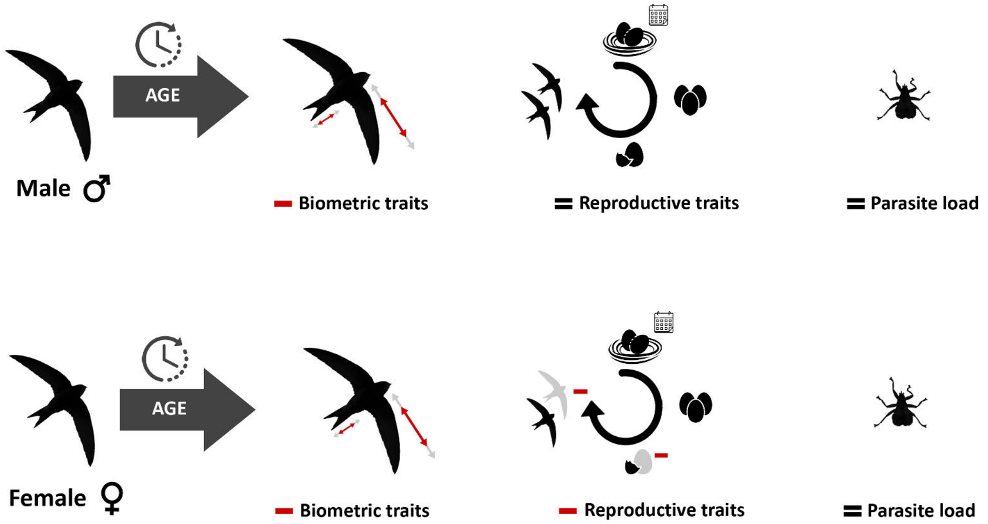

Figure 7. Schematic summary of the results of **Chapter I.**

4.2 Parental age effects

In **Chapter I**, I observed that both male and female Alpine swifts exhibit senescence in multiple traits. In **Chapter II**, I investigated the consequences of this senescence for the next generation by studying the effect of parental age at conception as a transgenerational factor affecting offspring phenotype and life-history. Specifically, I studied the short- and long-term effects of maternal and paternal age at conception on both male and female offspring. In the Asian elephant, effects of maternal age at birth on offspring life history have already been addressed by Reichert et al., (2020), which is why I focused my analyses on the Alpine swift.

4.2.1 Short-term effects on offspring phenotype

Because early life conditions are known to affect life-history trajectories in adulthood (Douhard et al., 2014; Festa-Bianchet et al., 2000; Hamel et al., 2009; Vasilieva & Tchabovsky, 2020), I examined parental age effects on offspring phenotype early in life, before fledging, which may provide insights into the mechanisms underlying later-life consequences of parental age. As observed in **Chapter I**, male and female Alpine swifts exhibit senescence in multiple traits. Older parents might thus also experience an age-related change in behaviour, and decline in parental care efficiency as a result of senescence (Catry et al., 2006; Hammers et al., 2021; Lecomte et al., 2010; Zimmer et al., 2011), potentially leading to reduced nestling condition, size, or immunity, as reported in the Barn swallow (Saino et al., 2002). Contrary to this prediction, parental age had a positive effect on nestlings before fledging: older parents produced larger offspring (with longer wings and tail) that were also less infested by ectoparasites, except sons from older fathers. Offspring body mass, however, was not affected by parental age at conception. This result aligns with a previous study showing that wing length and thus feather growth best reflect the cumulative effects of environmental conditions over the nestling developmental period, whereas body mass rather captures the short-term effect of environmental conditions on energy reserves (Masoero et al., 2024). The positive effect of parental age on offspring early-life phenotype likely results from enhanced parental care provided by older parents, potentially due to greater rearing experience and improved foraging efficiency (Cameron et al., 2000; Forslund & Pärt, 1995b; Weladji et al., 2006).

Daughters and sons from older mothers, and daughters from older fathers, had a lower parasite load. Parasite removal appears inefficient in the Alpine swift and is thus unlikely to explain these results. Instead, these parental age effects on parasite load are more plausibly linked to physiological mechanisms influencing nestling immunity. Offspring of older parents (except sons from older fathers) may have better immunity, resulting from better parental care and greater food provisioning provided by their parents. This would be consistent with previous studies, such as on the Barn swallow and Blue-footed booby (*Sula nebouxii*), which found an effect of parental care (Saino et al., 1997) and maternal age (Beamonte-Barrientos et al., 2010; Saino et al., 2002) on offspring immunity. In the Alpine swift, offspring receiving better parental care from older parents could mount a better cutaneous immune response, making the biting site unfavourable for the parasite, damaging their tissue (Owen et al., 2010; Wikel, 1996, 1999), and affecting their blood meal size and survival (Bize, Jeanneret, et al., 2008).

In **Chapter I**, I found evidence of reproductive senescence of females, characterised by a decline of brood size at hatching and fledging. This pattern likely reflects physiological senescence, resulting in lower egg quality and reduced hatching success among older mothers. In contrast, results from **Chapter II** showed that older mothers and fathers produced larger offspring with lower parasite loads, thus of higher quality before fledging. Although hatching success may decrease with maternal age due to a decline in reproductive physiology (Bize, Devevey, et al., 2008), increasing experience in chick rearing may compensate for this decline, enabling older parents to rear larger fledglings with fewer parasites. Additionally, the offspring from older mothers may benefit from a favourable growing environment within a smaller brood, thus with fewer siblings, more parental care per chick, and lower sibling competition.

4.2.2 Long-term effects on offspring life-history

I found that paternal age negatively influenced the lifespan of their sons and tended to reduce their LRS. In contrast, neither maternal nor paternal age influenced offspring age at first reproduction. The paternal-age effect observed in the Alpine swift parallels patterns reported in another long-lived bird, the Common tern, where older fathers produce sons with shorter lifespans and LRS (Bouwhuis et al., 2015). In this species, authors also found an effect of maternal age, older mothers producing daughters with reduced annual reproductive success, leading to lower LRS. However, the results vary across bird species. In the Seychelles warbler (*Acrocephalus sechellensis*), older mothers produce daughters with shorter lifespans and lower LRS (Sparks, Hammers, et al., 2022). In the House sparrow, older fathers produce sons with reduced annual reproductive success and LRS, while older mothers produce daughters showing the same pattern (Schroeder et al., 2015). In the Alpine swift, the reduced LRS of sons from older fathers was driven primarily by their shorter lifespan and, consequently, shorter breeding period. Similar patterns are reported in the Common tern and Seychelles warbler, where parental age effects on offspring LRS appear to stem from shorter breeding lifespans (Bouwhuis et al., 2015; Sparks, Hammers, et al., 2022). In the Alpine swift, this result is consistent with the fact that, in this species, LRS is largely determined by variation in adult lifespan (Mourocq et al., 2016; Tettamanti et al., 2012). This pattern seems consistent across long-lived species, such as most seabirds (Moreno, 2003). In contrast, in shorter-lived species such as the Great tit (*Parus major*) and House sparrow, offspring LRS is more closely linked to their reproductive productivity (Bouwhuis et al., 2010; Schroeder et al., 2015). Along the fast-slow continuum of life histories26/03/2026 15:58:00, long-lived species typically produce fewer offspring annually; long-lived birds, for instance, lay smaller clutches (Haukioja & Hakala, 1979; Liu et al., 2023; Zammuto, 1986), but invest more heavily in each reproductive attempt to maximise recruitment. Therefore, in slow-living species, individual differences tend to centre on survival and lifespan, whereas in fast-living species, variation is more pronounced in reproductive traits, such as annual breeding success (Van de Walle et al., 2023). As a result, in long-lived species, individuals that live longer have more breeding opportunities and thus contribute more to the future gene pool, and while their reproductive performance also improves with experience as they age, overall leading to higher LRS (Moreno, 2003). Therefore, increasing parental age at conception can reduce offspring fitness by shortening their lifespan, especially in long-lived species.

Since offspring quality improved with both maternal and paternal age, the shorter lifespan of sons from older fathers is unlikely to result from reduced paternal care received early in life. This assumption is supported by results from an experimental cross-fostering study on wild house sparrows, showing that offspring fitness was not influenced by the age of their foster parents (Schroeder et al., 2015). The negative effect of paternal age on sons' lifespan (i.e., the Lansing effect; Lansing, 1947) is therefore more likely driven by underlying physiological mechanisms and epigenetic transmissions from fathers to sons (see *4.5 Underlying mechanisms of sex differences*). Furthermore, because sons of older fathers tended to be more heavily infested by ectoparasites before fledging, their lower lifespan could also stem from a reduced immune capacity.

Finally, as no evidence of selective disappearance was detected, the parental age effects observed were not driven by demographic changes within the population.

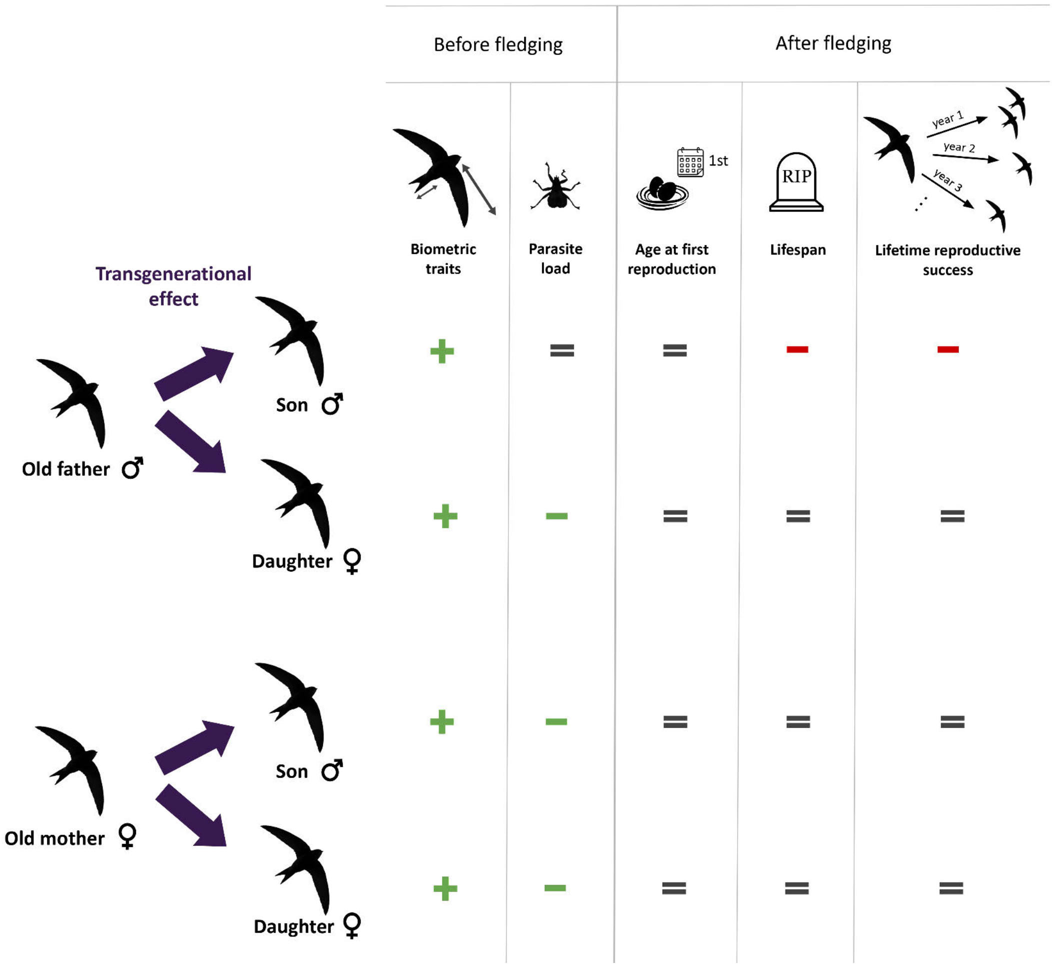

Figure 8. Schematic summary of the results of **Chapter II.** +: Positive effect, −: negative effect, =: no effect.

4.3 Differences between males and females

4.3.1 Sex-specific onset and rate of senescence

In **Chapter I**, the results revealed sex-specific ageing trajectories, and different onset and rate of senescence between males and females. Interestingly, females exhibited clear reproductive senescence, whereas males did not. Instead, males showed significant reproductive improvement in their last reproductive attempt. One explanation may lie in sex differences in investment between reproduction and self-maintenance. Although both sexes share parental care (incubate the eggs, feed the offspring, and potentially display similar levels of self-feeding, as evidenced in the closely related common swift;

Martins & Wright, 1993), older males and females could change their life history strategies with age. For example, male wandering albatross show a decline in reproductive performance later in life, as they would invest more in self-maintenance than reproduction (Lecomte et al., 2010). In the Alpine swift, our results suggest that older males might change their strategy in investing more in reproduction later in life, as indicated by the increase in brood size at hatching during their final breeding attempt. This strategic change may occur at the expense of self-maintenance, as older males showed decreased body mass and increased fork fluctuating asymmetry at their last reproduction. Such a pattern would support the hypothesis of a trade-off between reproduction and survival (Stearns, 1992). Interestingly, the findings from **Chapter II** further support the idea of late-life reproductive investment, as old-age fathers produced offspring with longer wings and tails. Again, this investment may come at the cost of self-maintenance, including somatic but also germline deterioration, which in turn would impact the life-history of the next generation, and explain the shorter lifespan and lower immunity in sons from old-age fathers.

4.3.2 Influence of sexual selection

Another potential explanation for the sex-specific reproductive senescence is attributable to sexual selection. Although Alpine swifts are socially monogamous, their divorce rate is relatively high (16.6% of pairs; Dumas et al., 2025), providing individuals with the opportunity to select different partners over the course of their lives. Males might preferentially mate with females that invest more in reproduction and/or have high reproductive success, or alternatively, females might prefer mating with males that are more resistant to the effects of ageing (Promislow, 2003). Because assortative mating by age is low in our Alpine swift population (Pearson r = 0.24), older males may also pair with younger females that have not started to senesce. In **Chapter II**, males began reproducing later than females, indicated by a significant effect of sex on offspring's age at first reproduction (see Table 1 in Moullec et al., 2025, **Chapter II**). This suggests that older males are mating with younger females, supporting the idea of sexual selection, from females to older, more experienced males or males to younger non-senescent females. However, detailed studies of sexual selection in the Alpine swift are still needed to understand how it can underlie the sex-specific patterns of senescence observed.

In addition to potential sexual selection with age, a phylogenetic comparative analysis found that fork depth in swifts would be under sexual selection (Hasegawa and Arai, 2020). Consistent with this, in **Chapter I**, I found a selective appearance effect in our population: males with longer and symmetrical fork started reproducing earlier. This indicates that fork length and symmetry may signal male quality, further supporting the hypothesis that male fork length is under sexual selection. Although no evidence of selective disappearance was detected for fork length, males with longer tails exhibited shorter lifespans than those with shorter tails. It might therefore be interesting to explore potential trade-offs between males' survival and investment in traits potentially under sexual selection (tail and fork length, fork fluctuating asymmetry). Interestingly, whereas AFR showed no significant influence on male reproductive performance, females that reproduced earlier had higher reproductive success (**Chapter I**). Moreover, females with higher body mass tended to live longer. Therefore, unlike males, which may face trade-offs among traits, high-quality females appear to perform better overall. Hence, there might also be a sexual selection from males towards good-quality females. Therefore, all things considered, it would now be necessary to investigate the connection between sexual selection (males towards younger and higher-quality females and females towards males with longer fork and tail), the relative investment of both sexes in parental care, and potential trade-offs between reproduction and survival, to better understand the sex-specific patterns of senescence observed.

4.3.3 Sex-specific transgenerational factors

In **Chapter II,** I found that both parental and offspring sex played a key role in shaping the observed transgenerational effects. The most striking sex-specific pattern was the paternal age effect on sons' lifespan and parasite load. Because the Alpine swift shows only cryptic sexual dimorphism (Dumas, Bize, et al., 2024; Dumas, St. Lawrence, et al., 2024), it is unlikely that parents can discriminate the sex of their offspring and adjust their care accordingly. Thus, these sex-specific transgenerational effects are more likely driven by physiological and epigenetic mechanisms that differ between sons and daughters (see *4.5 Underlying mechanisms of sex differences*).

4.4 Additive genetic variance and heritability

4.4.1 Evidence of evolutionary potential of lifespan

In **Chapter III**, I investigated whether genetic factors influence the inter-individual variation in lifespan and survival in the MTE Asian elephant population. Although when testing all individuals combined, the additive genetic variance and heritability of lifespan were close to zero, as often found in natural populations (Kruuk et al., 2000; McCleery et al., 2004; Coltman et al., 2005; Schroeder et al., 2012; Vedder et al., 2022), I found substantial additive genetic variance with the sex-specific model and thus substantial heritability values in both sexes. From the rare studies on wild populations reporting additive genetic variance of lifespan for males and females separately, some found estimates close to 0 in both sexes (Coltman et al., 2005; Kruuk, 2004; McCleery et al., 2004; Poissant et al., 2016), whereas a study on collared flycatchers (Merilä & Sheldon, 2000) and another on red-billed gulls (Teplitsky et al., 2009) found substantial estimates in males and close to 0 in females. The substantial V_A for lifespan found in both sexes in Asian elephants suggests an evolutionary potential of lifespan in this population. In addition, this result demonstrates that additive genetic variance can be maintained in natural populations of long-lived species. In the MTE population, additive genetic variance might have been maintained through the management practices of the population. Elephants are often moved between camps, and during their free time, they can mate with wild conspecifics. This might keep a continuous gene flow between the semi-captive and wild elephants throughout the country, and may lead to the low inbreeding, absence of local genetic drift, and high genetic diversity reported in this population (Maurer et al., 2024).

4.4.2 Sex-specific genetic factors

With the sex-specific model, I also found a low cross-sex genetic correlation, which likely contributes to sex-specific lifespan in the population. Cross-sex genetic correlation is a key parameter for understanding the evolutionary dynamics of a trait and its differences between sexes (Brommer et al., 2007; Kirkpatrick, 2009; Kruuk et al., 2008). The low cross-sex genetic correlation found in the Asian elephant population indicates that genetic factors influencing lifespan in one sex only weakly affect lifespan in the other sex, leaving potential for sex-specific evolution of longevity in response to selection. This cross-sex genetic correlation for lifespan is one of the few ever reported in the literature in vertebrates (Poissant et al., 2010), and more broadly adds to the rare values on fitness-related traits (Kruuk et al., 2008). Due to the scarcity of values reported for lifespan, it is difficult to discuss the value found for Asian elephants in the light of other populations. One study, on wild great tits (*Parus major*), reported the cross-sex genetic correlation for reproductive lifespan and found a negative value ($r_A = -0.430$, 95% CI: −0.859, 0.584; Poissant et al., 2016). In addition, a study on wild red deer found no cross-sex genetic correlation of survival (Foerster et al., 2007). The few results thus appear contrasted across populations. Overall, the low estimate found for lifespan in Asian elephants is consistent with values reported in the literature on fitness-related traits, suggesting a low cross-sex genetic correlation of fitness components relative to other traits such as morphology, behaviour, and development (Poissant et al., 2010).

4.4.3 Age-specific genetic factors

To understand the genetic basis of a trait, it appears crucial to also investigate how genetic parameters vary with age (Kruuk et al., 2008). Results from the age-specific model suggest a variation of genetic parameters with age in the Asian elephant population. Additive genetic variance was substantial between 0-5 years old, leading to a relatively high heritability for early-life survival (h^2=0.33). The other age categories, 6-19 and 20+ years, showed estimates close to 0. In MTE elephants, calves between 0-5 years old display a high mortality rate with a peak before 1 year old (Figure 3). Although in this population the taming process increases calves' mortality (Crawley et al., 2020; Mar et al., 2012), the high additive genetic variance in the first years of life indicates the influence of intrinsic factors, thus genetic effects, on survival variation compared to later in life (after 5 years old), when extrinsic causes appear as a main cause of mortality (Mar, 2007). For instance, some calves may carry deleterious mutations or alleles that reduce their survival, leading to premature death before age five. These harmful genetic factors would subsequently be removed from the population, after the death of the calves, resulting in lower genetic variation influencing survival in the older age categories. Two studies on primates found similarly high additive genetic variance of survival/mortality early in life. In captive western lowland gorillas (*Gorilla gorilla gorilla*), additive genetic variance and heritability of infant mortality were high before 1 year and then declined until 2 years (Ahsan & Blomquist, 2015). In captive female rhesus macaques (*Macaca mulatta*), the heritability of infant survival was high from birth to 30 days but declined close to zero from 30 days to 1 year (Gagliardi et al., 2010). More generally, some studies in other wild vertebrates and humans also found a variation (increase or decrease depending on the trait) of additive genetic variance and/or heritability with age, such as in body mass, morphological, and reproductive traits (Charmantier et al., 2005; Korkeila et al., 1991; Pettay et al., 2008; Réale et al., 1999; Wheelwright et al., 2014). Such age-specific genetic parameters might thus be widespread among traits and populations. Yet, quantitative genetic studies examining the age-specific genetic variation of traits remain limited.

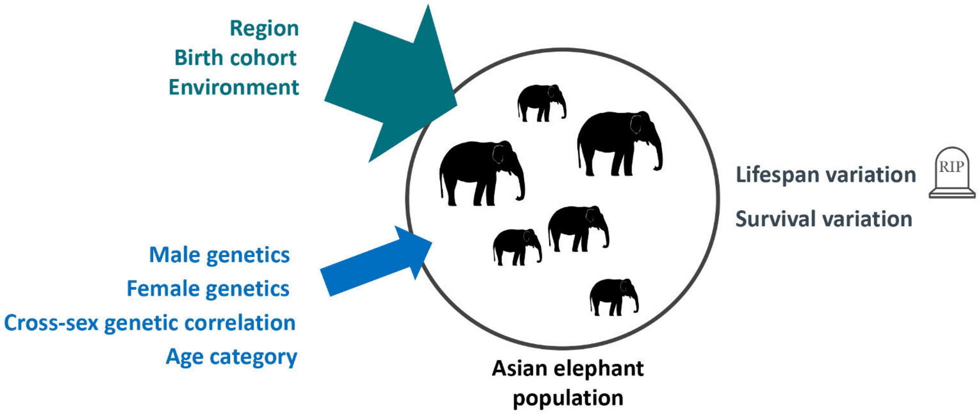

Figure 9. Schematic summary of the results of **Chapter III.**

4.5 Underlying mechanisms of sex differences

4.5.1 Sex-specific sensibility to environmental conditions

The mechanisms underlying individual differences in senescence and lifespan, such as differences between males and females as evidenced in this thesis, are complex. An important factor described in the literature is the sex-specific response to environmental conditions, especially early in life, which has been widely evidenced (Jones et al., 2009; Wilkin & Sheldon, 2009). Three hypotheses were proposed to explain this sex-specific sensitivity (Lindström, 1999). Sexual size dimorphism and thus energy requirements, usually higher in males, might disadvantage them and affect their survival more in the event of bad environmental conditions, such as low food availability. Although this hypothesis supports the lower survival and lifespan of male Asian elephants, a species with important sexual dimorphism, in the Alpine swift, males have longer lifespans than females, and they exhibit only low sexual dimorphism (Dumas, Bize, et al., 2024; Dumas, St. Lawrence, et al., 2024). It would thus be needed to investigate males versus females' survival during harsh environmental conditions in Alpine swifts, to see if the hypothesis holds in this species. Second, the heterogametic sex may exhibit greater susceptibility to the effects of deleterious recessive alleles located on the sex chromosomes. Because mammals and birds differ in their sex chromosome structure, with males being the heterogametic sex (XY) in mammals and females being the heterogametic sex (ZW) in birds, the heterogametic sex hypothesis aligns with the sex difference in lifespan in both the Asian elephant and the Alpine swift. Third, the higher level of androgen hormone needed in males for sexual development might antagonistically affect other physiological systems, which would decrease male performance as opposed to females in an unfavourable environment. Although this hypothesis is supported in Asian elephants, it does not hold for the Alpine swift, which exhibits longer lifespans in males and low sexual dimorphism. (Dumas, Bize, et al., 2024; Dumas, St. Lawrence, et al., 2024). Moreover, when experimentally parasitised, female offspring show greater mortality than males in the Alpine swift, which does not support the idea of a higher sensitivity of males to the early environment (Bize et al., 2005).

4.5.2 Underlying mechanisms of sex-specific senescence

In addition to the mechanisms described above, some specific mechanisms related to the Alpine swift might explain the sex-specific reproductive senescence observed in **Chapter I**. An important factor may be the differences between males and females in their reproductive physiology. As opposed to males, females bear important costs from the high energy expenditure of egg production. Such reproductive costs, reviewed by Williams (2005), can lead, for example, to decreased egg quality and rearing efficiency as observed in lesser black-backed gulls (*Larus fuscus*) (Monaghan et al., 1998; Nager et al., 2000). As previously mentioned, oocyte quality and egg composition in the Alpine swift might thus decrease in ageing females. On the contrary, as no senescence of reproductive performance was evidenced in males, it is unlikely that sperm quality and fertility decrease with age, contrary to observations in Barn swallows (Møller et al., 2009). In females, the deregulation of the pituitary-hypothalamic-ovarian axis in response to somatic senescence can also play a key role in their reproductive senescence (Lemaître & Gaillard, 2017). Alternatively, trade-offs may arise from changes in energy allocation strategies, as mentioned previously. Females might allocate more resources to somatic functions later in life, leading to a decline in reproductive function, thus reproductive senescence, whereas the opposite strategy might be adopted in males. In addition, a previous study on the Great tit and Alpine swift found evidence of senescence at the cellular level, with a decline in cell resistance to oxidative stress, and an association between oxidative stress resistance and survival (Bize et al., 2014). Interestingly, a higher resistance to oxidative stress is associated with increased survival in males and bigger clutch size in females (Bize, Devevey, et al., 2008). Hence, at the cellular level, a variation in the resistance to oxidative stress with age may contribute to the sex-specific patterns of senescence observed.

4.5.3 Underlying mechanisms of sex-specific parental age effects

The mechanisms by which older parents have a negative effect on offspring fitness, as observed in **Chapter II**, remain little understood, but there is an increasing number of studies addressing this question. Recent studies suggest that sex-specific parental age effects on offspring fitness traits may be driven by age-related deterioration of the parental germline and associated epigenetic transmission processes (Bouwhuis et al., 2015; Monaghan & Metcalfe, 2019; Schroeder et al., 2015). It is now well established that, in addition to the soma, the germline deteriorates with age in both males and females, which can reduce the fertility in older parents and decrease offspring quality (Monaghan & Metcalfe, 2019; Preston et al., 2015). Such age-related alterations of the germline can result in the non-genetic inheritance of factors that affect offspring fitness, including in birds, and these effects can be sex-specific (Bonduriansky et al., 2012; Bonduriansky & Day, 2009; Guerrero-Bosagna et al., 2018; Vigé et al., 2008). Three mechanisms associated with germline deterioration were reviewed by Monaghan & Metcalfe (2019): the accumulation of mutations, telomere attrition, and increasing mitochondrial dysfunction in gametes with age.

As individuals age, unrepaired mutations can accumulate in both male and female germlines and, once transmitted to the next generation, may impair offspring health, for example, by increasing disease susceptibility (Kong et al., 2012; Monaghan & Metcalfe, 2019; Paul & Robaire, 2013). Most of the studies investigating germline mutations, thus far mainly restricted to humans and mice, indicate that these mutations are more common in male than female germ cells (Ellegren, 2006; Monaghan & Metcalfe, 2019). The transmission of age-related accumulated mutations is therefore a plausible mechanism underlying the paternal age effect on sons' lifespan observed in the Alpine swift. Mitochondrial dysfunction also increases with age in female oocytes as a result of oxidative damage and the accumulation of mutations in mitochondrial DNA (Monaghan & Metcalfe, 2019). Because mitochondria are maternally inherited, the transmission of dysfunctional mitochondria from older mothers constitutes a potential mechanism explaining maternal age effects on offspring lifespan (Monaghan & Metcalfe, 2019; Wilding, 2015), as observed, for example, in the Asian elephant population (Reichert et al., 2020), but not in the Alpine swift population. Telomere length, a biomarker of ageing known to be related to individuals' fitness, appears as another strong candidate mechanism underlying the paternal age effects on sons' lifespan identified in **Chapter II**. In the alpine swift, telomere length and the rate of telomere loss were found to predict life expectancy (Bize et al., 2009), consistent with findings in other bird species (e.g. Bauch et al., 2014; Haussmann et al., 2005; Heidinger et al., 2012; Salomons et al., 2009). Studies found that telomere length is heritable (Broer et al., 2013), and its inheritance can be sex-specific, as demonstrated in lizards (Olsson et al., 2011) and several bird species (e.g. Asghar et al., 2015; Horn et al., 2011; Reichert et al., 2015). Across several species investigated, studies report a negative paternal age effect on offspring telomere length (Bauch et al., 2019; Bouwhuis et al., 2018; De Frutos Benítez et al., 2016; Eisenberg, 2019; Morosinotto et al., 2022; Noguera et al., 2018; Olsson et al., 2011; Sparks, Spurgin, et al., 2022; but see Bennett et al., 2022). This pattern is further supported by an experimental study on the Alpine swift showing that offspring of older fathers carry shorter telomeres (Criscuolo et al., 2017). Older fathers may therefore have shorter telomeres, including in their germline, presumably due to repeated cell division and accumulated damage, which are then transmitted to their offspring, potentially affecting their sons' lifespan. Finally, alteration in DNA methylation, a well-recognized biological marker of aging (Horvath & Raj, 2018), may similarly be transmitted across generations through the germline. Experimental studies in laboratory mice showed that the sperm of older fathers have altered DNA methylation, which is transmitted to their offspring. These offspring display altered DNA methylation, shorter lifespan, increased age-related pathologies, and altered gene expression and behaviour (Milekic et al., 2015; Xie et al., 2018). In the Alpine swift, these potential mechanisms remain to be tested, including whether they influence lifespan through reduced survival, accelerated senescence, or compromised immunity, as suggested by the results of **Chapter II**.

4.5.4 Importance of genetic factors in sex-specific lifespan

In the Asian elephant, females live longer than males and exhibit higher survival rates throughout their lives (Figure 3; see also Lahdenperä et al., 2018), supporting the common observation in mammals. The results of **Chapter III** suggest that this difference in lifespan between sexes is partly genetically determined. The low cross-sex genetic correlation for lifespan indicates that genetic factors that influence lifespan in one sex only weakly affect longevity in the other sex, leaving potential for sex-specific evolution of lifespan in response to selection. This sex-specific genetic architecture might be due to sex-dependent genetic factors influencing lifespan in males and females, as evidenced in recent studies (Bou Sleiman et al., 2022; Yuan et al., 2020). Additionally, sex-specific genetic factors or gene expression influencing traits related to survival, such as growth, immune function, or metabolic regulation, can lead to indirect, sex-biased genetic architectures for lifespan. For example, empirical studies have evidenced sex differences in immune gene expression (Klein & Flanagan, 2016).

Second, males and females have distinct reproductive characteristics which may generate sex-specific selection pressures on lifespan and contribute to the low cross-sex genetic correlation observed (Poissant et al., 2010). In Asian elephants, males body size and social rank increase with age (Chapman et al., 2016; Lalande et al., 2022; Lee & Moss, 1995; Lindeque & Jaarsveld, 1993; Poole, 1989; Sukumar, 2003), leading to a right-skewed reproductive success relative to females, that peaks later in life, as reported in African elephants (Moss, 1983; Poole, 1989; Rasmussen, 2005; Sukumar, 2003). In contrast, female reproductive output peaks at around 20 years of age and declines after approximately 50 years, often followed by a post-reproductive period (Chapman et al., 2019; Lahdenperä et al., 2014). Despite reproductive cessation, females can still contribute to their fitness by providing help to raise their grandchildren, also known as the grandmother effect (Lahdenperä et al., 2004, 2016). Grandmothers, as well as other female kin such as aunts and sisters, may provide care and sometimes allosuckling for the calves (Lahdenperä et al., 2016; Lee, 1987; Lynch et al., 2019). This cooperative behaviour may increase the fitness of older, non-reproductive females while reducing the reproductive costs borne by mothers. Taken together, males tend to achieve higher reproductive success later in life, whereas female reproduction peaks earlier and may be followed by a post-reproductive period during which fitness can be maintained through allomaternal care. These contrasting reproductive characteristics likely result in different lifespan optima for males and females and, consequently, distinct selection pressures acting on lifespan in each sex. Such sex-specific selection may favour different genetic variants in males and females, reducing the shared additive genetic variance between sexes and thereby lowering the cross-sex additive genetic variance estimate (Connallon & Matthews, 2019; Lande, 1980; Poissant et al., 2010). As a result, lifespan may evolve partly independently in males and females, contributing to the observed sex differences in lifespan in this species.

4.6 Environmental factors affect ageing

4.6.1 Early-life environmental conditions affect ageing in the Alpine swift

Because the individuals examined in this thesis live under natural conditions, environmental factors are expected to play a major role in shaping ageing trajectories and lifespan. This expectation was supported by the findings of **Chapters II** and **III**, which revealed clear environmental effects on both lifespan and survival. In **Chapter II**, I found both short- and long-term effects of early-life environmental conditions. Offspring born a year with high nestling mortality across the colonies, corresponding to a harsh environment during the breeding season, were significantly smaller (smaller wing, tail, and sternum length) before fledging, yet exhibited longer lifespans. High nestling mortality may be the consequence of unfavourable climatic conditions. Because Alpine swifts are insectivorous, they face food shortages under prolonged periods of rain and cold weather, which strongly constrain nestling growth (Bize et al., 2006). Similarly, I found that individuals reared in a larger brood, indicative of a more competitive environment and higher nestling mortality (Bize & Roulin, 2006), were significantly smaller and had a lower mass at 50 days, mirroring results of an experimental study on zebra finches (De Kogel, 1997). Offspring raised under such adverse conditions, whether due to increased sibling competition or reduced access to food and nutrients, may thus experience slower growth, which may explain their smaller size before fledging (Masoero et al., 2024).

In addition, the longer lifespan of offspring born a year with high nestling mortality may result from this slower growth rate, consistent with the ‘growth rate-lifespan trade-off hypothesis’, which has been experimentally demonstrated in the literature (Lee et al., 2013; Metcalfe & Monaghan, 2003). Alternatively, such harsh early-life conditions might have imposed a selection for higher quality offspring, with higher survival rates, and thus longer lifespan, following the “viability selection hypothesis” (Hadfield, 2008; Mojica & Kelly, 2010). An ongoing study on the same bird population is investigating the long-term consequences of harsh early-life environmental conditions, and the results appear consistent with the “viability selection hypothesis” (Jarayseh et al., 2025).

4.6.2 Environmental conditions affect lifespan and survival in the Asian elephant

In **Chapter III**, environmental factors explained a substantial proportion of the variation in lifespan and survival in the Asian elephant population. Both the geographic region and birth cohort, representing the spatial and temporal environmental conditions experienced by the elephants, accounted for a large share of the lifespan and survival variance. Because MTE elephants are exposed to ecological conditions similar to those faced by fully wild populations, they exhibit comparable survival rates and fecundity (Clubb et al., 2008, 2009). Seasonal fluctuations in Myanmar, particularly in precipitation, temperature, and food availability across the hot, cold, and monsoon seasons, have been shown to strongly influence the health and physiological state of the elephants. Previous studies found that such seasonal variation affects health parameters, testosterone levels, stress levels, and survival of MTE elephants (dos Santos et al., 2020; Moullec et al., 2024; Mumby et al., 2013b, 2013a; Ukonaho et al., 2023). Furthermore, the pronounced birth cohort effects highlight the importance of early-life environmental conditions, reinforcing the idea that experiences during the early developmental period can have lasting consequences for individual life-history trajectories. Overall, **Chapters II** and **III** demonstrate the importance of extrinsic factors, both during early life and in adulthood, in shaping lifespan and survival.

4.7 Strengths and limits of the thesis

4.7.1 Strengths of the thesis

Addressing the critical issue of why and how individuals age has key demographic and health consequences. Yet, ageing research has primarily focused on captive and short-lived genetically homogenous animal models (Hurria et al., 2019), which limits our understanding of how ageing unfolds in nature. In contrast to such lab models, wild animals exhibit important genetic variation, live in fluctuating environments, and are exposed to a variety of stressors. Studying senescence in the wild, however, is difficult because it requires long-term longitudinal data collected from large numbers of individuals of known age that live long enough to observe signs of senescence (Nussey et al., 2008). Consequently, detailed, long-term data are rare for wild, long-lived populations, although they are essential for gaining new insights into senescence in nature, its underlying mechanisms, and consequences for fitness. Moreover, most studies on senescence focus on survival and reproductive traits (Bouwhuis & Vedder, 2017b; Lemaître, Ronget, Tidière, et al., 2020; Lemaître & Gaillard, 2017; Nussey et al., 2013), and are often limited to reproduction in females (Nussey et al., 2013), which may hinder a comprehensive understanding of senescence within and across species (Lemaître & Gaillard, 2017). In this thesis, I have access to two populations of long-lived species, the Asian elephant and the Alpine swift, with long-term longitudinal data on individuals followed throughout their whole lives. The main strength of this thesis lies in the combination of data created by this project, rarely available for long-lived species. This data covers a wide range of phenotypic and life-history traits: biometric traits, reproductive traits, parasite burden, survival, and lifespan, with information on both male and female individuals. Therefore, this thesis benefits from an exceptional study setting to investigate ageing, lifespan, and the genetic and transgenerational factors affecting them. In addition, this thesis provides new insights into ageing in species from two different taxa. These two species are complementary. On the one hand, we have an extremely long-lived mammal with a longevity similar to that of humans that can exceed 70 years (Chapman et al., 2019). This provides information on ageing factors on a very long time scale. On the other hand, we have a long-lived bird that can reach 26 years of age (Tettamanti et al., 2012). These two species have very distinct physiology, reproductive characteristics, and thus life-histories, and live in two very different environments, exposing them to contrasting ecological stressors. Therefore, studying ageing in these two taxa expands our understanding of the diversity of ageing patterns and will help disentangle ageing processes that are taxon-specific from those that are shared across taxa in the wild. Finally, this thesis uses an integrative approach to study ageing, incorporating population-level variation among individuals, sex-specific differences, environmental influences, and both transgenerational and genetic effects. This multifactorial framework provides a more comprehensive understanding of the mechanisms shaping senescence and lifespan in nature. Overall, by combining exceptionally detailed long-term datasets with this integrative analytical perspective, this thesis provides valuable insights into our understanding of ageing in the wild and paves the way for future studies of key importance in evolutionary biology.

4.7.2 Limitations of the thesis

The different chapters of this thesis inevitably come with certain limitations. Although the thesis benefits from large datasets, the number of old individuals in **Chapter I** and old parents in **Chapter II** remains relatively small. In **Chapter I**, estimating senescence rates required pooling males and females into a single model, and individuals with unknown age at first reproduction had to be retained, preventing me from testing how

AFR influences the onset and rate of senescence. In **Chapter II**, the limited number of old-age mothers and fathers reduced statistical power and thus the precision of the results. To address this, I evaluated the robustness of all findings using sensitivity analyses and adopted a cautious approach when discussing the results, distinguishing the most robust findings from those that should be interpreted more carefully (see Table 3 from Moullec et al., 2025, **Chapter II**). In **Chapter III**, due to the uncertainty of birth dates before 1960, I restricted the analyses to elephants born after that date, which excluded the oldest individuals from the dataset. Although Asian elephants, including those in the MTE population, can live beyond 70 years (Chapman et al., 2019), the final dataset contained individuals with a maximum lifespan of "only" 59 years (Figure 3). Consequently, the study lacks representation of the longest-lived fraction of the population. This reduced sample size diminished the precision of estimates of V_A and h^2 for survival in the 20+ age category, resulting in a very wide confidence interval. Furthermore, because of the small sample size of these longer-lived individuals, it was not possible to test the heritability of survival for the longest living fraction of the population (>50 years old). Such analysis would have been particularly informative given that studies in humans report higher heritability of lifespan among the top 10% of survivors (van den Berg et al., 2019), increasing heritability with older age thresholds (Gögele et al., 2011), and a genetic basis underlying late-life survival (Christensen et al., 2006; Gudmundsson et al., 2000; Hjelmborg et al., 2006; Perls et al., 1998; Schoenmaker et al., 2006; van den Berg et al., 2019).

In **Chapters I** and **II**, another limitation is the absence of quantified information on the investment of both parents in parental care (e.g., incubation, foraging, food provisioning, visit to the nest). Although males and females both provide parental care by incubating the eggs and feeding the nestlings, we do not have data on their respective contributions (e.g., time spent incubating, foraging, provisioning versus self-feeding), nor do we know whether parental care strategy changes with age as observed in the Wandering albatross (Lecomte et al., 2010). Similarly, information on sexual selection is lacking, particularly regarding mate choice based on reproductive performance, age, or biometric traits. Such behavioural information would be key to better understand the mechanisms shaping the sex-specific senescence patterns observed in **Chapter I**, as well as the sex-specific parental age effects on offspring traits described in **Chapter II**. A deeper investigation of sexual selection processes in the Alpine swift is therefore needed.

In **Chapters I** and **II**, we also lack information on the number of recruits to be able to quantify the actual fitness costs of senescence in both sexes (i.e., the parental age effect and the age-related variation in the number of recruits produced by males and females). Unfortunately, such data cannot be obtained for our population of Alpine swifts. After fledging, we only have records for individuals that subsequently began breeding in the colonies that are monitored.

In **Chapter III**, the main limitation arises from the absence of paternal connections in the pedigree. Without information on paternity, the model has difficulty disentangling additive genetic effects from maternal effects, leading to potential confounding between the two. This lack of paternal pedigree information likely contributes to the apparent absence of maternal effects in our models, although, in mammals, maternal effects typically have a strong influence on early-life survival and offspring phenotype (Cheverud & Wolf, 2009). For example, substantial maternal effects have been reported for longevity in red deer (*Cervus elaphus*; Kruuk et al. 2000), and for infant mortality in captive western lowland gorillas (*Gorilla gorilla gorilla*; Ahsan and Blomquist 2015). While some studies suggest that incomplete pedigrees generally lead to an underestimation of additive genetic variance (Cantet et al., 2000; Dong et al., 1988; Wolak & Reid, 2017), Morrissey et al. (2007) demonstrated that missing paternal information can instead inflate estimates of additive genetic variance while underestimating maternal effects. Consequently, the absence of paternal links may have inflated the additive genetic variance estimates for longevity and, in particular, for early-life survival (0-5 years), thus from birth until calves are separated from their mother, when maternal effects are expected to be most important (Cheverud & Wolf, 2009).

In **Chapter I**, it could have been informative to model the ageing trajectory of survival and thus determine the onset and rate of actuarial senescence. This would allow evaluating whether the senescence of reproductive and biometric traits is a potential consequence of actuarial senescence, or vice versa, and identify potential trade-offs between these traits. More specifically, we could observe whether the absence of reproductive senescence in males occurs at the expense of an early and strong decline in survival, and whether females show the opposite pattern, thus testing the disposable soma theory of ageing. Such additional analyses would help to unravel which traits and trade-offs are responsible for the fitness costs of senescence, an issue of particular importance in the evolutionary biology of ageing. Similarly, in **Chapter II**, it would have been interesting to test the effect of parental age on offspring annual survival. This would help disentangle whether the lower lifespan of sons from older fathers is a result of a lower survival throughout life or a higher rate of senescence later in life.

Finally, in **Chapter III**, it would be highly valuable to measure the heritability and additive genetic variance of fitness (i.e., number of recruits produced in the lifetime of an individual), and other fitness components such as annual survival, to better understand the rate of adaptive evolution in the MTE elephant population (Bonnet et al., 2022). However, in our dataset, components of fitness such as the lifetime reproductive success were too imprecise to be investigated due to the high number of censored individuals, thus with unknown age at death and incomplete reproduction.

4.8 Future research direction

In Chapter **III**, I found evidence that both genetic and environmental factors contribute to variation in lifespan in the elephant population. Likewise, both genetics and the environment are known to shape senescence rates. Importantly, as pointed out by Monaghan et al., (2008), it is important to distinguish between lifespan and senescence rates, as well as the factors influencing each. Therefore, the next step would be to investigate the heritability of the onset and rate of senescence to determine whether the observed patterns are mediated by genetic factors. Similarly, it would be needed to assess the factors affecting the onset and rate of senescence. In light of the results from **Chapters I** and **II**, it would be valuable to examine how environmental conditions, parental age at conception, and reproductive performance, both early in life and during adulthood, affect the onsets and rates of senescence observed in the Alpine swift.

For **Chapters I** and **II**, additional physiological analyses would be necessary to deepen our understanding of the observed results. A key follow-up study would involve collecting blood and plasma samples to measure immunity and ageing biomarkers (e.g., telomere length, mitochondrial function, oxidative stress, DNA methylation) and to examine their relationship with the patterns of senescence and parental age effects identified. Ultimately, such analyses would help unravel the underlying mechanisms at play. In addition, given the strong influence of environmental conditions on ageing and lifespan highlighted throughout this thesis, it would be interesting to investigate in more detail the effect of local climate. One approach would be to integrate data from meteorological institutes (i.e., temperature, precipitation) in Switzerland for the Alpine swift and in Myanmar for the Asian elephant, to assess how climatic conditions experienced early in life and at adulthood shape ageing trajectories and lifespan in these two long-lived species. Taken together, investigating the variation with age of physiological factors (immunity, biomarkers of ageing) in interaction with fine-scale information of environmental data, to investigate their interplay in shaping senescence patterns, within and among species, would be highly valuable and remains largely unexplored (Gaillard & Lemaître, 2020).

Finally, a logical next step would be to compare ageing patterns across traits previously modelled in the Asian elephant to assess the asynchrony of senescence in this species, and to estimate the heritability of lifespan and survival in the Alpine swift. This would enable direct comparisons between the two species and help determine whether similar patterns emerge across these distinct taxa. Such a comparative framework would generate valuable metrics for future meta-analyses on senescence and lifespan evolution, thereby enhancing our understanding of the evolutionary mechanisms shaping senescence in nature.

5 Conclusion

In **Chapter I**, I showed that multiple traits in the Alpine swift, including biometric and reproductive traits, vary with age and show senescence later in life. Importantly, I found sex differences in senescence patterns and an asynchrony of senescence across traits within individuals. The difference in ageing patterns between males and females may be explained by mating choices (based on age or performance), sex-specific life-history strategies such as allocation to reproduction versus self-maintenance, and by sex-specific physiological and genetic factors. Within individuals, the asynchrony between traits may reflect differences in energetic costs (e.g., laying date versus brood size at fledging in females), differences in the strength of selection among traits (e.g., wing length versus tail length for flight performance), or trade-offs between traits (e.g., reproduction versus somatic maintenance in males).

In **Chapter II**, I found that transgenerational effects linked to parental age at conception contribute to ageing and lifespan diversity in the Alpine swift. Older fathers produced sons with shorter lifespans and higher parasite load before fledging, suggesting a sex-specific transmission of non-genetic factors from fathers to sons. The senescence documented in **Chapter I** likely extends to the germline (e.g., telomere shortening in germ cells), meaning that deteriorated germ cells in old fathers may transmit detrimental epigenetic or cellular factors that influence sons' longevity and immune function. Parental age also positively affected offspring quality early in life, probably through increased breeding experience.

In **Chapter III**, I showed that part of the variance in lifespan was explained by heritable genetic factors in both sexes. I also found a low cross-sex genetic correlation, suggesting that genes affecting lifespan in one sex have only weak effects in the other, or that males and females experience selection pressures that favour different lifespan optima. In addition, the additive genetic variance and heritability of survival varied with age, being substantial early in life (0-5 years) but declining thereafter. These findings demonstrate an evolutionary potential for lifespan and early-life survival in a natural population of a long-lived mammal.

Finally, extrinsic factors from the environment play a key role in shaping ageing diversity, as observed through **Chapters II** and **III**. In the Alpine swift, individuals exposed to an adverse early-life environment, characterised by high nestling mortality, lived longer. In the Asian elephant population, spatial and temporal variation in environmental conditions (i.e., region and birth cohort) explained a substantial part of the variance in lifespan. Environmental effects on lifespan likely occur through the modulation of life-history trade-offs, such as between growth rate and survival, or through impacts on physiological mechanisms influencing individuals' survival.

In conclusion, this thesis demonstrates that ageing diversity in natural populations of long-lived vertebrates is shaped by the combined effects of genetic factors, sex, parental age, life-history strategies, and environmental conditions experienced both early in life and at adulthood.

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